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Manual on meat inspection for developing countries

by
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in cooperation with
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Introduction

Meat inspection is commonly perceived as the sanitary control of slaughter animals and meat. The aim of meat inspection is to provide safe and wholesome meat for human consumption. The responsibility for achieving this objective lies primarily with the relevant public health authorities who are represented by veterinarians and meat inspectors at the abattoir stage.

In many developing regions and in particular in rural abattoirs, meat inspectors often lack the necessary information and guidelines to assess the sanitary status of carcasses, meat and organs from slaughter animals. FAO has therefore endeavoured to prepare concise guidelines on the subject together with colour illustrations demonstrating the pathological lesions that may occur in bovines, small ruminants, pigs, game, poultry and rabbits. The statements made on the judgement of diseased carcasses or parts of the carcasses are recommendations which are also influenced by the need of salvaging as much meat as possible for human consumption. These recommendations are not meant to interfere with any existing regulations on the subject in individual countries.

This Manual on Meat Inspection for Developing Countries has been prepared by an experienced meat inspection specialist as the main author in cooperation with meat inspection experts from the four regions Asia and Pacific, Africa, Latin America and the Near East. The book is intended to guide meat inspectors particularly in the four mentioned regions in their daily work in urban and rural abattoirs. Veterinarians engaged in meat inspection will also benefit, especially as regards their supervisory roles in meat hygiene. The book shall also serve as a training manual for trainees in meat inspection, a field in which FAO has organized theoretical and practical training courses for many years. FAO will continue these activities in future and it is expected that the Manual will facilitate these tasks.

The Code of Hygienic Practice for Fresh Meat and the Codes for Anti-Mortem and Post-Mortem Inspection of Slaughter Animals published recently by the Joint FAO/WHO Codex Alimentarius Commission is a useful supplement to this publication and provides additional information on meat hygiene and inspection procedures.

CHAPTER 1

MEAT INSPECTION PROCEDURES

The objectives of meat inspection programme are twofold:

a. To ensure that only apparently healthy, physiologically normal animals are slaughtered for human consumption and that abnormal animals are separated and dealt with accordingly.

b. To ensure that meat from animals is free from disease, wholesome and of no risk to human health.

These objectives are achieved by antemortem and postmortem inspection procedures and by hygienic dressing with minimum contamination. Whenever appropriate the Hazard Analysis Critical Control Point (HACCP) principles should be used: The inspection procedures should be appropriate to the spectrum and prevalence of diseases and defects present in the particular class of livestock being inspected using the principles of risk assessment.

ANTEMORTEM AND POST MORTEM INSPECTION OF FOOD ANIMALS GENERAL PRINCIPLES

Antemortem Inspection

Some of the *major objectives* of antemortem inspection are as follows:

- to screen all animals destined to slaughter.

- to ensure that animals are properly rested and that proper clinical information, which will assist in the disease diagnosis and judgement, is obtained.

- to reduce contamination on the killing floor by separating the dirty animals and condemning the diseased animals if required by regulation.

- to ensure that injured animals or those with pain and suffering receive emergency slaughter and that animals are treated humanely.

- to identify reportable animal diseases to prevent killing floor contamination.

- to identify sick animals and those treated with antibiotics, chemotherapeutic agents, insecticides and pesticides.

- to require and ensure the cleaning and disinfection of trucks used to transport livestock.

Both sides of an animal should be examined at rest and in motion. Antemortem examination should be done within 24 hours of slaughter and repeated if slaughter has been delayed over a day.

Spread hogs and animals affected with extensive bruising or fractures require emergency slaughter. Animals showing clinical signs of disease should be held for veterinary examination and judgement. They are treated as "suspects" and should be segregated from the healthy animals. The disease and management history should be recorded and reported on an *A/M* inspection card. Other information should include:

1. Owner's name
2. The number of animals in the lot and arrival time

3. Species and sex of the animal
4. The time and date of antemortem inspection
5. Clinical signs and body temperature if relevant
6. Reason why the animal was held
7. Signature of inspector

Antemortem inspection should be carried out in adequate lighting where the animals can be observed both collectively and individually at rest and motion. The general behaviour of animals should be observed, as well as their nutritional status, cleanliness, signs of diseases and abnormalities. Some of the abnormalities which are checked on antemortem examination include:

1. Abnormalities in respiration
2. Abnormalities in behaviour
3. Abnormalities in gait
4. Abnormalities in posture
5. Abnormalities in structure and conformation
6. Abnormal discharges or protrusions from body openings
7. Abnormal colour
8. Abnormal odour

Abnormalities in respiration commonly refer to frequency of respiration. If the breathing pattern is different from normal the animal should be segregated as a suspect.

Abnormalities in behaviour are manifested by one or more of the following signs:

The animal may be:

a. walking in circles or show an abnormal gait or posture
b. pushing its head against a wall
c. charging at various objects and acting aggressively
d. showing a dull and anxious expression in the eyes

An *abnormal gait* in an animal is associated with pain in the

legs, chest or abdomen or is an indication of nervous disease.

Abnormal posture in an animal is observed as tucked up abdomen or the animal may stand with an extended head and stretched out feet. The animal may also be laying and have its head turned along its side. When it is unable to rise, it is often called a "downer". Downer animals should be handled with caution in order to prevent further suffering.

Abnormalities in structure (conformation) are manifested by:

a. swellings (abscesses) seen commonly in swine
b. enlarged joints
c. umbilical swelling (hernia or omphalophlebitis)
d. enlarged sensitive udder indicative of mastitis
e. enlarged jaw ("lumpy jaw")
f. bloated abdomen

Some examples of *abnormal discharges* or protrusions from the body are:

a. discharges from the nose, excessive saliva from the mouth, afterbirth
b. protruding from the vulva, intestine
c. protruding from the rectum (prolapsed rectum) or uterus
d. protruding from the vagina (prolapsed uterus)
e. growths on the eye and bloody diarrhoea

Abnormal colour such as black areas on horses and swine, red areas on light coloured skin (inflammation), dark blue areas on the skin or udder (gangrene).

An *abnormal odour* is difficult to detect on routine A/M examination. The odour of an abscess, a medicinal odour, stinkweed odour or an acetone odour of ketosis may be observed.

Since many abattoirs in developing countries have not

accommodation station or yards for animals, Inspector's antemortem judgement must be performed at the admission of slaughter animals.

Postmortem inspection

Routine postmortem examination of a carcass should be carried out as soon as possible after the completion of dressing in order to detect any abnormalities so that products only conditionally fit for human consumption are not passed as food. All organs and carcass portions should be kept together and correlated for inspection before they are removed from the slaughter floor.

Postmortem inspection should provide necessary information for the scientific evaluation of pathological lesions pertinent to the wholesomeness of meat. Professional and technical knowledge must be fully utilized by:

1. *viewing, incision, palpation* and *olfaction techniques.*

2. *classifying the lesions* into one of two major categories - *acute or chronic.*

3. establishing whether the *condition is localized or generalized*, and the extent of systemic changes in other organs or tissues.

4. *determing the significance of primary and systemic pathological lesions* and their relevance to major organs and systems, particularly the liver, kidneys, heart, spleen and lymphatic system.

5. *coordinating all the components of antemortem and postmortem findings* to make a final diagnosis.

6. *submitting the samples to the laboratory for diagnostic support*, if abattoir has holding and refrigeration facilities for carcasses under detention.

Carcass judgement

Trimming or condemnation may involve:

1. Any portion of a carcass or a carcass that is *abnormal* or *diseased*.

2. Any portion of a carcass or a carcass affected with a condition that may present a *hazard to human health*.

3. Any portion of a carcass or a carcass that may be *repulsive to the consumer*.

Localized versus generalized conditions

It is important to differentiate between a localized or a generalized condition in the judgement judgement of an animal carcass. In a *localized* condition, a lesion is restricted by the animal defense mechanisms to a certain area or organ. Systemic changes associated with a localized condition may also occur. Example: jaundice caused by liver infection or toxaemia following pyometra (abscess in the uterus).

In a *generalized* condition, the animal's defense mechanisms are unable to stop the spread of the disease process by way of the circulatory or lymphatic systems. The lymph nodes of the carcass should be examined if pathological lesions are generalized. Some of the signs of a generalized disease are:

1. Generalized inflammation of lymph nodes including the lymph nodes of the head, viscera and/or the lymph nodes of the carcass

2. Inflammation of joints

3. Lesions in different organs including liver, spleen kidneys and heart

4. The presence of multiple abscesses in different portions of the carcass including the spine of ruminants

Generalized lesions usually require more severe judgement than localized lesions.

Acute versus chronic conditions

Acute conditions

An acute condition implies that a lesion has developed over a period of some days, whereas a chronic condition implies the development of lesions over a period of some weeks, months or years. A subacute condition refers to a time period between an acute and chronic condition.

The acute stage is manifested by inflammation of different organs or tissues, enlarged haemorrhagic lymph nodes and often by petechial haemorrhage of the mucosal and serous membranes and different organs such as heart, kidney and liver. An acute stage parallels with the generalized disease complex, when an acute infection tends to overcome the animal's immune system and becomes generalized.

Each case showing systemic lesions should be assessed

individually taking into account the significance that these lesions have towards major organ systems, especially the liver, kidneys, heart, spleen and lymphatic system as well as the general condition of the carcass.

Chronic conditions

In a chronic condition, inflammation associated with congestion is replaced by adhesions, necrotic and fibrotic tissue or abscesses. The judgement in the chronic stage is less severe and frequently the removal of affected portions is required without the condemnation of the carcass. However, judgement on the animal or carcass judgement tends to be more complicated in subchronic and sometimes in peracute stages. If generalized necrotic tissue is associated with previous infection, carcass must be condemned.

GUIDELINES FOR MINIMUM POSTMORTEM INSPECTION REQUIREMENTS (CATTLE, HORSES, SHEEP & GOATS, PIGS AND GAME)

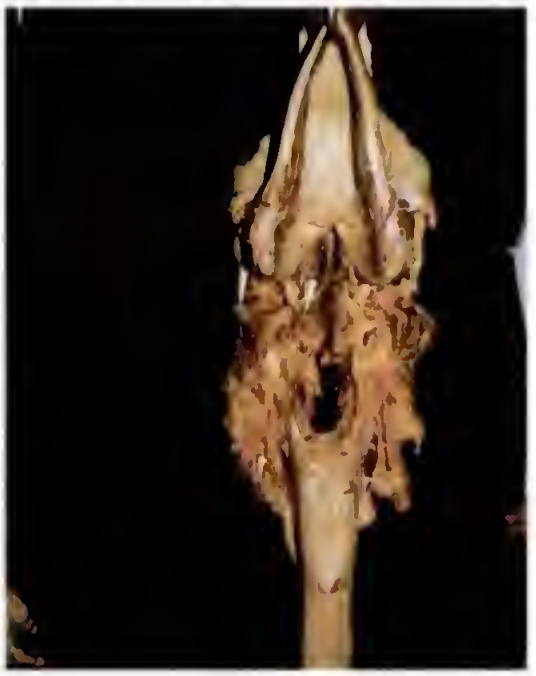
HEADS

General View external surfaces. For cattle, horses, pigs and game view the oral and nasal cavities.

Lymph nodes (Fig. 1)

Submaxillary	Incise(a)
Parotid	Incise(a)
Retropharyngeal	Incise(a)

View and incise by multiple incision or slicing.



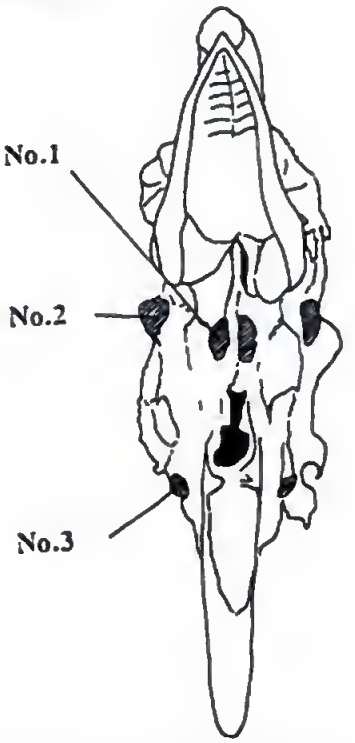


Fig. 1 : Head inspection. Retropharyngeal (No.1), parotid (No.2) and submaxillary (No.3) lymph nodes are viewed and incised by multiple incisions and slicing.

Fig. 2: Head inspection in buffalo. Retropharyngeal lymph nodes (No.1) are viewed and incised by multiple incisions and slicing.

Tongue View and palpate (view only in calves up to 6 weeks of age).

Other **Cattle** - except in calves up to six week of age, the oesophagus of all cattle and calves should be separated from its attachment to the trachea and viewed.
- as part of inspection of all cattle and calves over the age of 6 weeks for Cysticercus bovis, the muscles of mastication should be viewed and one or more linear incisions made parallel to the lower jaw into the external and internal muscles of mastication; in addition one incision into M.triceps brachii, 5 cm behind the elbow, should be made.

Horse - the head should be split lengthwise in the medial line and the nasal septum removed and examined in all horses that are from areas where glanders is endemic.

Pigs - where there is a risk of Cysticercus cellulosae being present, the outer muscles of mastication, the abdominal and diaphragmatic muscles and the root of the tongue of all pigs should be incised and the blade of the tongue viewed and palpated;

Game - inspection cuts for tapeworm cysts are not necessary, as these cysts are generally not infective for humans.

NOTES

- These are guidelines for inspection requirements, the inspection can be made more intensive or less intensive depending on the outcome of the examination.

- "incise" means multiple incisions or slicing.

- "palpate" as used above means to view and palpate.

GUIDELINES FOR MINIMUM POSTMORTEM INSPECTION REQUIREMENTS (CATTLE, HORSES, SHEEP & GOATS, PIGS AND GAME)

VISCERA

Lungs (Fig. 3)

View and palpate. Except in sheep and goats, the bronchi should be opened up by a transverse incision across the diaphragmatic lobes. For horses and cattle, the larynx, trachea and main bronchi should be opened along their length.

Lymph nodes

Bronchial (tracheobronchial) and mediastinal: Incise, (a) (see "Notes")

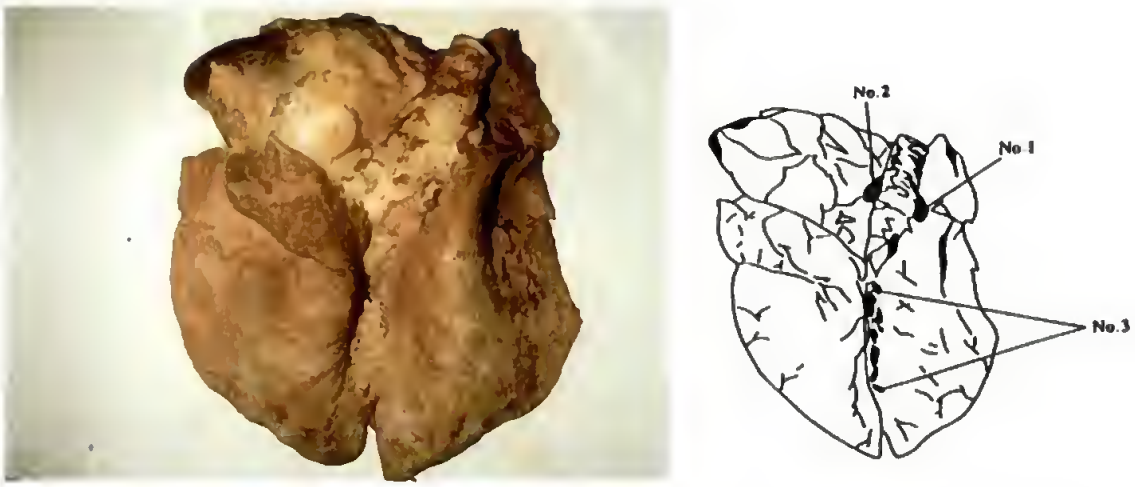


Fig. 3: Lung inspection - Bronchial left (No.1) and right (No.2) and mediastinal (No.3) lymph nodes are viewed and incised.

Fig. 4: Lung inspection in buffalo - Open trachea and incised bronchial and mediastinal lymph nodes.

Heart (Fig. 5)

View after the removal of the pericardium. Additional inspection requirements for cattle as per (b).

Additional inspection requirements for pigs as per (c).

Fig. 5: Heart inspection - Lengthwise incisions (minimum four) from base to apex into the heart muscles. Observe cut surfaces.

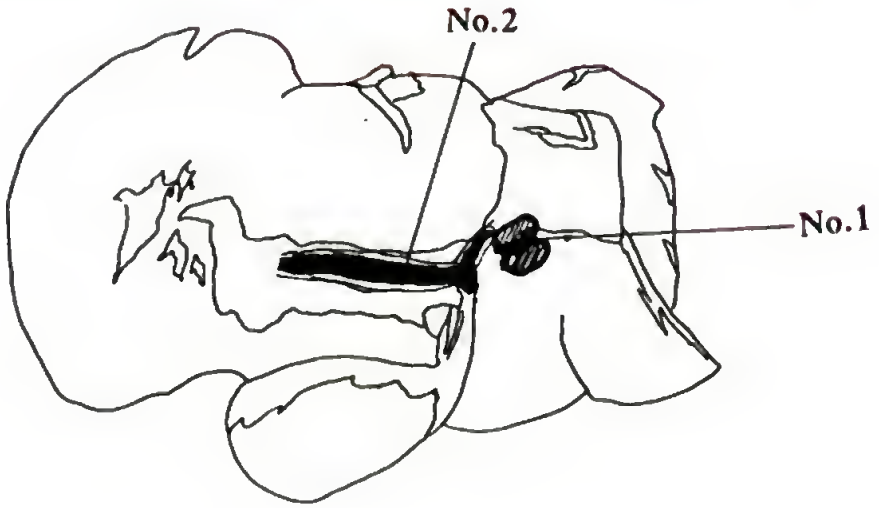
Liver (Fig. 6)

View and palpate entire surface(both sides). View the gall bladder. For cattle over 6 weeks of age, incise as deemed appropriate to detect liver flukes. Open large bile ducts. For sheep, pigs and game, incise as deemed appropriate for parasite.

Lymph nodes

Portal (hepatic), view and incise

Fig. 6: Liver inspection - Incised portal (hepatic) lymph nodes (No.1) and opened large bile duct (No.2).



Spleen (Fig. 7)

Palpate

Fig. 7: Stomachs and spleen inspection - Viewing of rumen and viewing and palpation of spleen.

Gastrointestinal tract (Fig. 8)

View (a)

Mesenteric lymph nodes (Fig. 9), View (a,d)

Fig. 8: Viewing of rumen, reticulum, omasum and abomasum.

Fig. 9: Viewing and incision of the mesenteric lymph nodes. In this case an incision was performed to demonstrate the mesenteric lymph nodes chain.

Kidneys

View after enucleation. In grey and white horses - Incise.

Uterus (adults), View

NOTES

- These are guidelines for inspection requirements, the inspection can be made more intensive or less intensive depending on the outcome of the examination.

- "incise" means multiple incisions or slicing.

- "palpate" as used above means to view and palpate.

- (a) view only in calves up to 6 weeks of age.

- (b) the heart of all cattle and calves over the age of 6 weeks should be inspected for Cysticercus bovis either by making one or more incisions from base to apex or by everting the heart and making shallow incisions that enable the cardiac valves and muscle tissue to be inspected; this inspection of the heart should also be undertaken in calves up to 6 weeks of age that are from areas where Cysticercus bovis is endemic.

- (c) the heart of all pigs derived from areas where there is a risk of Cysticercus cellulosae being present, should be opened up and the deep incision made into the septum.

- (d) incise if any lesion were observed in the submaxillary lymph nodes.

GUIDELINES FOR MINIMUM POSTMORTEM INSPECTION REQUIREMENTS (CATTLE, HORSES, SHEEP & GOATS, PIGS AND GAME)

CARCASS

General

Examine carcasses (including musculature, exposed bones, joints, tendon sheaths etc.) to determine any signs of disease or defect. Attention should be paid to bodily condition, efficiency of bleeding, colour, condition of serous membranes (pleura and peritoneum), cleanliness and presence of any unusual odours.

Lymph nodes[1]

Superficial inguinal (male) (Fig. 10) - Palpate
Supramammary (female) - Palpate (a)
External and internal iliac (Fig. 10, Fig. 11) - Palpate (b)
Prepectoral (Fig. 12) - Palpate
Popliteal (Fig. 13) - Palpate (only sheep/goats and game/antelope)
Renal (Fig. 12) - Palpate (cattle, horses, pigs) or incise if diseases is suspected.
Prescapular (Fig. 14) & prefemoral - Palpate (only sheep and goats)

[1]In all animals in which systemic or generalized disease is suspected, in all animals positive to a diagnostic test for tuberculosis, in all animals in which lesions suggestive of tuberculosis are found at postmortem inspection, the main carcass lymph nodes being the precrural, popliteal, anal, superficial inguinal, ischiatic, internal and external iliac, lumbar, renal, sternal, prepectoral, prescapular and atlantal nodes, as well as the lymph nodes of the head and viscera, should be incised and examined.

Other

The muscles and the lymph nodes (lymphonodi sub-rhomboidei) beneath one of the two scapular cartilages of all grey or white horses should be examined for melanosis after loosening the attachment of one shoulder.

NOTES

- These are guidelines for inspection requirements, the inspection can be made more intensive or less intensive depending on the outcome of the examination.

- "incise" means multiple incisions or slicing.

- "palpate" as used above means to view and palpate.

- (a) incise when udder is or has been in lactation or in case of mastitis.

- (b) means iliac nodes in pigs.

Fig. 10: Superficial inguinal and internal and external iliac lymph nodes in a pig. Viewed and palpated on routine P/M examination.

Fig. 11: Medial view of the hind quarter. Superficial inguinal, internal and external iliac and lumbar lymph nodes are palpated and incised in systemic or generalized disease.

Fig. 12: Medial view of the fore quarter with intercostal, suprasternal, presternal and prepectoral lymph nodes. Presternal and prepectoral lymph nodes are incised.

Fig. 13: Popliteal lymph nodes in a pig. These nodes are incised if a systemic or general disease is suspected.

Fig. 14: Lateral view of the carcass. Precrural and prescapular lymph nodes are incised in systemic or generalized disease.

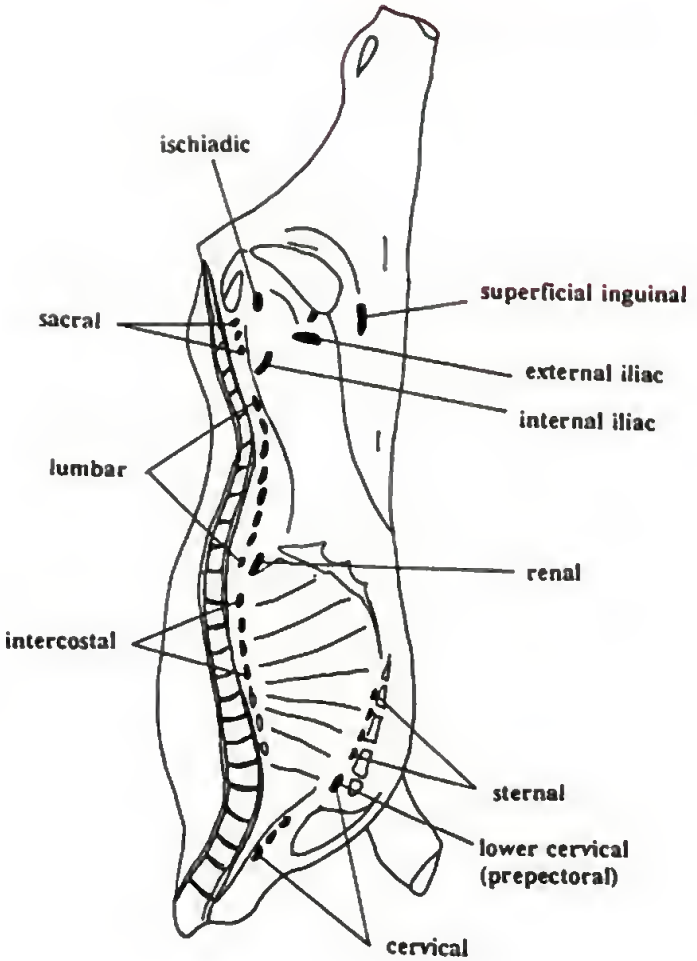


Fig. 15: Medial view of carcass with relevant lymph nodes

ANTEMORTEM AND POSTMORTEM INSPECTION OF POULTRY

Antemortem inspection of birds presents some difficulties if the birds are placed in crates or liners, and hence only a superficial inspection of their general condition is carried out. The remainder of the poultry examination should take place after the birds have been hung in shackles and before they are bled. The records of antemortem inspection are mandatory and should include date and time of inspection, truck number, species, the total number of birds and the name of the owner. The objectives of antemortem examination are:

- to determine the *general condition of the birds*

- to establish if *a disease or condition requires particular handling* such as segregation of diseased birds, delayed slaughter or adjustment of line speed.

In inclement weather, particular in winter, birds require immediate slaughter. In the summer, the steady change of air in the truck or in the holding area should be maintained. In cases of reportable disease, such as avian influenza or Newcastle disease, a veterinarian should be informed and all pertinent information should be recorded. Some diseases have similar signs on *A/M inspection*. For example, infectious bronchitis may be confused with Newcastle disease. A differential diagnosis is required in such cases.

Postmortem inspection in poultry refers to inspection techniques and inspection of carcasses and viscera. P/M examination consist of viewing, palpation and smell. The colour, shape, and consistency of organs and tissues must be observed singly or in combination. The colour of the poultry carcass depends on age, sex, nutrition and the scalding temperature during slaughter.

Carcasses must be suspended at 2 or 3 points depending on the class of poultry. Intestinal tract, liver, spleen, and heart (viscera) must be exposed for visual examination and palpation. A poultry inspector (Fig. 16) should be able to look inside the carcass and detect any pathological lesions such as airsac inflammation, peritonitis, oviduct inflammation (salpingitis) etc. Contamination by faeces and bile should also be observed. During the examination of viscera and carcass, both hands should be used. External lesions on the carcass include the swelling of the sinuses, nasal and ocular discharge (if the head is present), skin lesions, joint swellings etc.

Judgement : Localized lesions could be disposed by an inspector, however the final judgement of the carcass should be done by a veterinarian. The condemnation of carcass is usually for pathological, non pathological and aesthetic reasons.

Fig. 16: Inspection of the viscera and carcass in a broiler.

SLAUGHTER AND INSPECTION OF GAME ANIMALS FOR MEAT

Some parts of the world continue to be blessed with large, thriving populations of game animals, in Africa particularly antelopes such as impala, kudu and eland, in the Southern part of Latin America hares and some deer and antelope species and in Eastern Europe red and roe deer. Controlled cropping of these herds can provide a significant, sustainable source of supplementary protein especially in rural areas.

In ideal circumstances and in case of the game meat is for export, two basic systems of culling and carcass preparation can be used.

1. The first system is one of night shooting on foot using spotlights. Animals which appear healthy are shot, immediately bled and the stomachs and intestine eviscerated on the spot. After a number of carcasses have been collected on an accompanying vehicle, they are then transferred to a central, permanent abattoir facility suitable for their dressing, inspection and refrigeration. Since antemortem inspection is performed by hunters, they should be trained in basic antemortem procedures in order that they may be able to select healthy from sick animals.

2. In the second system animals are rounded up and herded into a temporary, funnel like structure. The animals are rested and antemortem inspection is carried out much more objectively. Animals are then shot at point blank range, bled and eviscerated immediately and removed to a temporary butchery for dressing, inspection and refrigeration. Ante and postmortem inspection can be objectively carried out with this system, although hygiene could be somewhat compromised. A judicious combination of features from both these systems, which can be modified, can be

used to suit a variety of circumstances in the field.

Antemortem and postmortem inspection procedures

The inspection procedures that are most appropriate to any particular type of game animal or carcass will vary not only according to species, but also according to whatever other information may be available about the wildlife population from which they are harvested. Minimum inspection procedures as set out in the Joint FAO/WHO Codex Alimentarius Commission's Code of Hygienic Practice for Game, are useful commencing points in developing appropriate procedures.

Judgement categories

The decision at inspection is classed into the following categories of Judgement:

1. Approved as fit for human consumption.

When the inspection and any other information available has revealed no evidence of any unacceptable disease or defect, and if the dressing has been implemented in accordance with hygienic requirements, the game carcass and offal should be approved as fit for human consumption without restriction provided no animal health restrictions are otherwise applicable.

2. Totally unfit for human consumption.

The game animal and all offal should be condemned or otherwise disposed of for inedible purposes if:

a. they are hazardous for food handlers, consumers and other animals;

b. they show decomposition, extensive injury, swelling edema, emaciation or contamination;

c. they show signs consistent with natural death, death by trapping or a moribund state.

d. there are unacceptable deviations, from normal game meat, detectable by sensory means.

3. Partially condemned.

Where lesions are localized, affecting only part of the carcass or offal, the affected parts should be removed and the unaffected parts conditionally or unconditionally passed.

Conditions affecting antelope

The antelope is perhaps the most preferred and frequently hunted species of game animal in Africa for the specific purpose of providing meat for human consumption. The impala is the easiest of the antelope to cull on a large scale, although the eland is almost as easy to manage as some domestic cattle. Favourable features of antelope are their apparent good herd-health and lack of pathological and parasitic conditions found at meat inspection. Causes of condemnation of the carcass, meat and offal in impala in Africa seem to fall into two categories:

a. Management related
b. Disease related

Management related:

trauma - due to gunshot wounds.

contamination - chiefly gross dirt attained from the environment during bleeding and de-gutting or intestinal contents during careless evisceration.

spoilage and putrefaction - wastage for these reasons can be considerable in Africa if operations are carried out during summer daytime. These losses can be minimized if hunting and dressing are done during the winter months, at night and during lower ambient temperatures.

Disease related:

Parasites

"measles" - tapeworm cysts of various kinds have been found in game carcass such as impala, kudu, bushbuck, reedbuck, sable, wildebeest (gnu, antelope) and warthogs. The cysts vary in size from that of a pea to a golf ball and are often seen in the peritoneal cavity, loosely attached to the serosa, viscera or in the musculature. There are no special predilection sites of the muscular cysts. Routine inspection incisions for measles in domestic animals are of no value in determining presence or degree of infestation in game animals. Serosal affection can be successfully trimmed before release but muscular parasites make the carcass aesthetically unacceptable. In the latter carcass can be boiled or used for manufacturing purposes. These cysts do not seem to affect humans.

Sarcocysts - these are frequently seen in the skeletal muscle of impala (mostly microscopic however); the carcass may have to

be condemned if severely affected.

Stilesia - this tapeworm may be found in the liver of small antelope and seem to be widespread in Africa. Trimming is required.

Cooperoides hepatica - this is a small brown filarial worm which occurs coiled up in a cyst in the liver, most frequently in impala. It is often associated with stilesia. Trimming is required.

Cordophillus - a filarial worm found encysted in the heart muscle of kudu. 25% of these animals are affected. This parasite is occasionally found in other muscle and may also occur in the heart muscle of domestic cattle. Affected tissue should be trimmed.

Hydatid - these cysts have been seen in the lungs and livers of impala, zebra, giraffe and warthog. If slight infestation is present affected tissue should be trimmed.

Pathological conditions - the incidence of septicemic/bacteremic conditions and pneumonia were extremely low.

SLAUGHTER AND INSPECTION OF FARMED GAME

Red deer and fallow deer and some of the antelopes mentioned above are the main species which are farmed for meat production. In particular in New Zealand, but also in Europe and some other regions game farming has become an important source of supplying the domestic and export markets with venison.

Farmed game is in many cases slaughtered in special premises

and is therefore subject to antemortem and postmortem inspection. Game farmers are now experienced enough to arrange for live animal transports on trucks to the game slaughterhouses. These abattoirs have specific lairages, where the animals can be rested. Slaughter takes place by using captive bolt pistols for stunning and bleeding, deboning evisceration and carcass dressing is similar to cattle slaughter, however carcass splitting is usually not performed.

Antemortem and postmortem inspection procedures and conditions affecting the farmed game are similar to the situation described for wild game. However, residues in meat (veterinary drugs, pesticides), parasitic diseases or infectious diseases such as tuberculosis may pose major problems than it is the case in wild game.

SLAUGHTER AND INSPECTION OF OSTRICHES

The slaughter of farm ostriches is fast becoming a commercial enterprise and may provide an important source of lean, high-protein meat for human consumption. The slaughter and dressing procedures consist essentially of stunning, bleeding defeathering and dressing. These operations are carried out in separate rooms.

a. - Stunning. The bird is stunned electrically using 90 volts at 1.5 amperes for approximately 20 seconds.

- Bleeding. The neck and vessels are severed behind the jaw.

- De-feathering. This is done manually in order to avoid damage to the skin follicles.

b. - Dressing is done in a manner similar to that of small ruminants. Organs are eviscerated in one set.

Antemortem inspection :

The following are characteristics of apparently **healthy ostriches**:

1. Alert and inquisitive with a bright eye and erected neck; occasionally lowering and then raising head.

2. Walks with a springy gait and may sometimes be aggressive.

3. Pecks inquisitively at shiny objects.

4. Produces thick, white clear urine and firm faeces.

5. The feathers are fluffed up and the body appears well rounded. The tail is well perked.

The following are characteristics of **sick ostriches**:

1. Lethargic and drooping neck and wings. It may sit down frequently or become recumbent. This clinical sign may also be observed in the stressed bird.

2. The eyes are half closed

3. The mucosa of the mouth may be very congested; the ostriches peck at food but do not swallow.

4. The abdomen may sometimes be bloated and blue/purple.

5. The urine may be green or brown and the faeces fluid or pasty.

6. The feathers appear bedraggled; the wings and tail drop.

Postmortem inspection :

It will be necessary for the head, pluck (heart, pericardium, liver, spleen, and lung if possible), alimentary tract, genitalia and carcass (with neck and kidney) to be properly identified and presented separately for inspection. The ostrich, like other avian species is lacking an organized lymphatic system. Since many viral and bacterial infections tend to be of a generalized nature, sound and professional meat inspection examination and judgement of the birds and carcass is of great importance.

Lungs not removed during dressing procedures should be examined visually and by palpation in the thorax. To expose the lungs, two cuts above the lungs on the each side of the ribs should be made.

Head Visual examination of the mouth, palate, eyes, lips and sinuses for icterus, sinusitis, crusting of eyelids and thrush (oral Candida infection)

Pluck Lungs - visual and palpation for haemorrhage, edema and pneumonia.

Heart - visual and palpation for haemorrhages; expose valves for endocarditis.

Pericardium - visual, and incision if necessary; for pericarditis.

Liver - visual and palpation; incise if necessary; for icterus, discolouration, adhesions, degeneration, abscess, fibrosis, inflammation and toxic conditions

Spleen - visual and incision if necessary; for enlargement, haemorrhages and signs of febrile or septic conditions.

Kidney - visual and palpation; for haemorrhages, degeneration, urate crystals.

Intestinal tract

Oesophagus/proventriculus, gizzard - visual and palpation; for foreign body penetration, impaction, inflammation and ulceration and parasitic conditions (nematode-Libyostrongylus) in glands of proventriculus.

Small intestine - visual and palpation; impaction, volvulus, necrotic and catarrhal enteritis and small tapeworm (Houttynia).

Large intestine - visual and palpation for faecal impaction, stones, inflammation and nematode (Condiostomum).

Reproductive organs - visual for egg retention, rupture, prolapsed penis; Atrophic organs are found during non-breeding season.

Carcass

Visual inspection of external and internal carcass surfaces, limbs and joints. Observe for contamination, inadequate bleeding, bruising, haemorrhages, lacerations, fracture, dislocation, twisted legs, adhesions, icterus, arthritis, peritonitis, air sacculitis, abscesses (injection sites), foreign bodies.

Judgement

Carcass should be condemned if affected with any of the following: death from any cause other than slaughter, extensive bruising and haemorrhages, general contamination, putrefaction, emaciation, edema, icterus, septicemia, aspergillosis, toxoplasmosis, malignant or multiple tumours, leucosis, poisoning. The parts of the carcass which show localized lesions may be trimmed and the rest of the carcass would then be approved.

SUPERVISION OF HYGIENIC DRESSING OF CARCASSES

1. During dressing the carcass is *exposed to contamination* from:

a) Abattoir environment including implements used, and the hands of the operators. A variety of bacteria, fungi and yeasts are in the abattoir environment. Studies in abattoirs indicate that salmonella counts in the implements used may vary from 0 - 270 per cm^2 or more in each implement, depending on their regular cleaning and sanitation the scabbards having the highest numbers.

b) Hides of the animals
Hides are heavily contaminated parts and can reach up to 3×10^6 bacteria per cm^2 or more.

c) Stomach and gastrointestinal contents
Gastrointestinal contents have the heaviest load of microorganisms. Faeces contain up to 9.0×10^7 bacteria per gram, and various numbers of yeast and mould. The ruminal contents have only slightly lower numbers of micro-organisms.

2. Therefore, during meat inspection it is an important duty of the inspecting officer to ensure that:

a) the implements used during slaughtering, dressing and meat inspection are well sanitised periodically or whenever they are likely to be contaminated;

b) during cutting into the hide and exposure of the carcass, the external surface of the hide does not contact the carcass meat;

c) the viscera are not accidentally opened during the dressing procedures or during evisceration.

3. If a carcass or part is contaminated with faeces or visceral contents such areas should be trimmed off. The opened viscera would have to be separated from the rest of the carcass as quickly as possible.

4. The introduction of a Hazard Analysis Critical Control Point (HACCP) concept can be helpful to maintain high standards of slaughter and dressing hygiene based on an assessment of the risks to human and animal health.

HAZARD ANALYSIS CRITICAL CONTROL POINT (HACCP) CONCEPT IN MEAT INSPECTION

A specific HACCP concept tailored to each abattoir and the class of animal should be developed to ensure the most efficient and effective concept of sanitary control.

The introduction of specific HACCP concept involves the following:

a) identifying hygienic hazards
b) ranking these hazards
c) defining the critical limit
d) identifying the critical control points
e) recommending necessary control
f) record keeping
g) verification procedures to ensure efficiency
h) tests to ensure that the concept is working

The Hazard Analysis Critical Control Point (HACCP) Concept was introduced in the food industry in 1971 to ensure that there would be effective control of the quality of processed foods. The World Health Organization (WHO) recommends that this concept also be applied to Meat Inspection and Meat Hygiene in particular to control salmonellosis. It can also be used to reduce bacterial contamination during slaughtering and dressing and to ensure quality control in Meat Inspection.

Meat Inspection and Meat Hygiene shall make sure that meat and meat products are safe and wholesome for human consumption. The practise of meat inspection has gradually changed over the last three decades. The classical antemortem and postmortem procedures were designed to detect disease in an animal before slaughter and the lesions produced by the disease after slaughter respectively. This was done by the use of senses (organoleptic

tests) such as the use of touch (palpation), sight (inspection and observation), smell (gangrenous smell) and taste (only in cooked products). Zoonotic diseases, particularly tuberculosis received high priority. Laboratory tests were done to confirm the disease when necessary or as appropriate.

With the gradual reduction in the incidence of animal tuberculosis in many countries along with the development of intensive methods of animal husbandry and the widespread use of pesticides and veterinary drugs, new problems are emerging. These are associated with residues on one hand and increased human infections with zoonotic agents contaminating animal foods on the other. There appears to be a general trend worldwide, with a few exceptions where human Salmonella infections have nearly doubled during the last five year period and human Campylobacter infections have nearly tripled during the same period.

Other bacteria that are causing increasing concern as food contaminants are Yersinia spp. and Listeria spp. There is simultaneously a greater consumer expectation of a longer shelf life in the finished fresh meat product. All these factors suggest that in the practise of meat inspection, it would be advantageous to use the HACCP concept to identify the critical control points at which these bacterial groups and other spoilage organisms may contaminate the carcasses, so that appropriate action can be taken. The critical control points that have been identified for Salmonella contamination in red meats, and poultry are shown in Fig. 17 and Fig. 18. These are applicable to other major bacterial contaminants as well.

These figures show that during red meat production, major contamination occurs in the abattoir during skinning and evisceration, that some contamination could occur during transport, lairage and deboning and that the most effective control point is in the chiller. Therefore, it is absolutely essential for meat

inspectors to ensure that skinning and evisceration are done properly. The critical control points during the slaughter of poultry (Fig. 18) are picking and evisceration. In developing countries where these tasks are not automated, it is necessary to ensure that proper hygienic precautions are taken during each of these operations. In automated plants, the machinery for picking and evisceration would need to be sanitised regularly, in particular when birds from different sources are slaughtered.

Fig. 17: Flow diagram showing sources of contamination with Salmonella and Critical Control Points (CCP) in Red Meat Production.

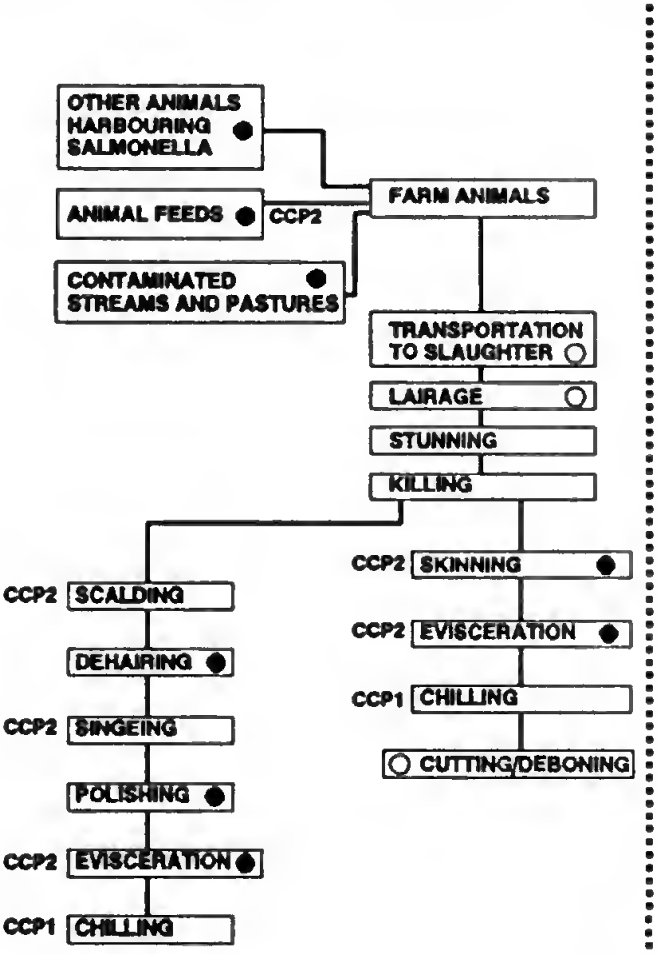
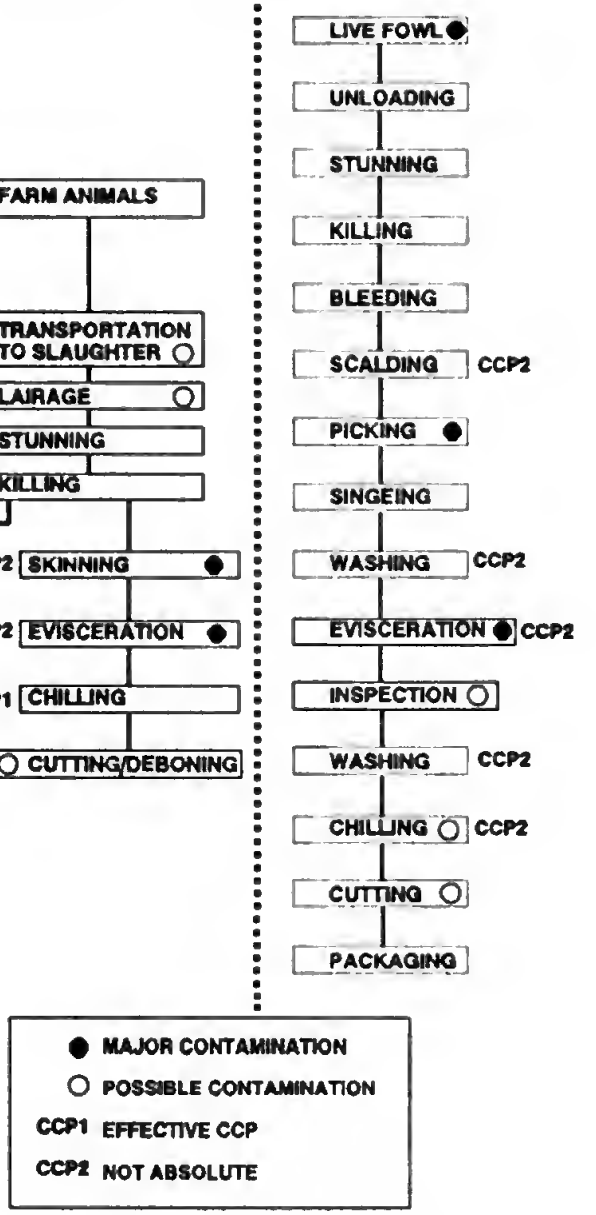


Fig. 18: Flow diagram showing sources of contamination with Salmonella and CCP in processing of Poultry Meat.



(Adapted from WHO 1986)

CHAPTER 2

GENERAL PATHOLOGICAL CONDITIONS

Fever (Pyrexia)

Fever is an abnormal elevated body temperature. It may be classified as septic and aseptic according to the presence or non presence of an infection. In *septic fever* the infection is caused by viruses, bacteria, bacterial toxins, protozoa and fungi. *Aseptic* fever may be caused by a) tissue necrosis as seen in muscle degradation due to intermuscular injection of necrotizing substances, in rapidly growing tumours undergoing necrosis or lysis of burned tissue; b) by chemicals or surgery. In former by an administration of drugs and in latter by breakdown of tissue and blood. c) during anaphylactic reaction of antibodies to the foreign antigens.

Antemortem findings :

1. Chills and sweating
2. Dehydration
3. Elevated body temperature
4. Increased pulse and respiration
5. Depression and dullness
6. Anorexia and obstipation

In septic fever the other signs may include

7. Diarrhoea and vomiting
8. Urinous or phenolic odour or breath
9. Shock, convulsions and coma

Postmortem findings :

1. Rigor mortis
2. Putrefaction
3. Congestion of subcutaneous blood vessels and carcass
4. Enlarged lymph nodes
5. Evidence of cloudy swelling of liver, heart and kidneys

Judgement : Carcass is *condemned* if fever syndrome is associated with presence of bacteria or bacterial toxins in the blood and/or findings of drugs and antimicrobial substances.

If typical signs of fevered carcass are not seen carcass should be held for 24 hours after slaughter and re-examined. In case of mild fevered syndrome detected first on postmortem inspection, the carcass may be *conditionally approved* with heat treatment providing that bacteriological and chemical test are negative.

Differential diagnosis : Hyperthermia and septicemia. In hyperthermia the elevation of body temperature is caused by physical factors such as high environmental temperature or prolonged muscular exertion, particularly in humid weather.

Inflammation in viral diseases

Inflammation associated with viral diseases is usually secondary to primary cellular change. Secondary bacterial infections frequently accompany and complicate viral diseases particularly respiratory and skin diseases. Viral infection associated with fever, malaise, anorexia or incoordination is attributed to absorption of injured cell products, viral toxicity and viral abnormalities which cause circulatory disturbances. Vascular shock together with viral toxicity and failure of one or more vital organs, is thought to be associated with death in viral diseases.

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Septicemia

Septicemia is a morbid condition caused by the presence of pathogenic bacteria and their associated toxins in the blood. The positive diagnosis of septicemia can only be made by isolation of the causative organism from the blood stream. This is not practised on routine antemortem examination of animals in abattoirs; however, the evidence of septicemia is determined by the antemortem and postmortem findings.

Antemortem findings :

1. Depression
2. Changes in body temperature. The temperature is usually elevated but it can also be normal and subnormal during the terminal phases.
3. Difficult and rapid breathing
4. Shivering and muscle tremors
5. Congestion or petechial haemorrhages of conjunctivae, mouth and vulvar mucosae

Postmortem findings :

1. Enlarged edematous or haemorrhagic lymph nodes
2. Degenerative changes in parenchymatous organs (liver, heart and kidneys)
3. Congestion and petechial or ecchymotic haemorrhages in kidney, heart surface, mucous and serous membranes, connective tissue and panniculus adiposis
4. Splenomegaly
5. Inadequately bled-out carcass as a result of high fever
6. Blood stained serous exudate in abdominal and/or thoracic cavities.
7. Anaemia resulting from bone marrow depression and icterus may also be present.

One or more lesions may be absent. However if one significant lesion is present, such as, generalized acute lymphadenitis, the carcass must be condemned. All gross lesions in the carcass and organs must be considered before the animal is judged septicemic.

Septicemia is found in many infectious diseases including acute forms of salmonellosis, leptospirosis, swine erysipelas, hog cholera and in anthrax in cattle.

Judgement : The animals, animal carcasses , offal and other detached portions of animals affected with septicemia are *condemned*. In borderline cases bacteriological examination should be done wherever possible.

Toxaemia

The identification of toxaemia presents some difficulties on routine antemortem and postmortem examination. The gross lesions differ depending on the specific organisms and toxins involved. Also the clinical signs of toxaemia simulate a variety of other pathologic conditions.

Toxaemia is defined as the presence and rapid proliferation of exotoxin and endotoxin derived from microorganisms or produced by body cells in the blood-stream. Clinical signs and postmortem findings are similar to those of septicemia.

Antemortem findings :

1. Normal or subnormal temperature. Fever may be present if toxaemia is due to microorganisms.
2. Confusion and convulsions
3. Abnormal changes in locomotion;
4. Moribund animal or evidence of pain (noted by grinding its

teeth).
5. Animal is not able to rise or rises with great difficulty
6. Dehydration may also be present

Postmortem findings :

1. Haemorrhage in organs
2. Normal or enlarged and edematous lymph nodes (not hyperplastic as in septicemia)
3. Areas of tissue necrosis
4. Emphysema in cattle
5. Rarely degenerative changes of parenchymatous organs (heart, liver and kidneys).

Toxaemia is frequently associated with:

6. Gangrenous mastitis
7. Metritis
8. Aspiration pneumonia
9. Old wounds and injuries
10. Diffuse peritonitis due to perforation of the reticulum or uterus.

All these signs may not be seen in every animal affected with toxaemia.

Judgement : If there is evidence of septicemia or toxaemia the carcass and the viscera should be *condemned* and the implements used during inspection and the hands and arms of the inspector should be washed and disinfected. The primary lesions causing septicemia or toxaemia including metritis, mastitis, pericarditis, enteritis and others, should be observed and recorded as causes of condemnation. Comatose or moribund animals should be condemned on antemortem examination.

Pigmentation

Pigments are classified as exogenous and endogenous. Exogenous pigments are synthesized outside of the body and endogenous within the body itself.

Pigments are coloured substances which accumulate in the body cells during the normal physiological process and abnormally in certain tumours and conditions. They have a different origins, biological significance, and chemical composition.

In anthracosis, the carbon particles are found as a black pigment in tissues. This condition is seen as black pigment of the lungs and corresponding lymph nodes in animals raised in urban areas. The lungs affected with anthracosis are condemned and the carcass is approved.

The carotenoid pigments are exogenous pigments, greenish-yellow in colour which consist of carotene A, carotene B, and xanthophyll. They are important in meat inspection because they cause yellowish discoloration in the fat and muscles of (Jersey and Guernsey) cattle. Carotenoid pigments should be differentiated from bile pigments in icterus. The bovine liver affected with this condition is enlarged and shows a bright yellow colour. Such a liver is condemned with the rationale that the affected liver demonstrates some toxic changes, as damaged liver cells cannot metabolize carotene. Liver carotenosis must be differentiated from pale livers in advanced pregnancy.

The endogenous pigments, except for melanin and lipofuscin are derivates of haemoglobin.

(A) Melanosis

Melanosis is an accumulation of melanin in various organs including the kidneys, heart, lungs and liver (Fig. 19), and other locations such as brain membranes, spinal cord, connective tissue, periosteum etc. Melanin is an endogenous brown-black pigment randomly distributed in tissue. In grey and white horses, this pigment is found under the shoulder, axillary area and ligamentum nuchae. Melanin is also found in lymph nodes, pig skin and belly fat or mammary tissue in female pigs. This condition is called "seedy belly" or "seedy cut" since the black colour in the mammary tissue resembles round, black seeds. The melanotic tissue in pigs shows a tendency towards neoplasia. Melanin deposits in the oesophagus and adrenal glands in older sheep are a common finding on postmortem examination. Multifocal deposits of melanin in the liver of a calf is known as "Melanosis maculosa". It is common in calves and it usually disappears after the first year of age.

Judgement : Carcasses showing extensive melanosis are *condemned*. If the condition is localized, only the *affected organ* or part of the carcass needs to be condemned.

Differential diagnosis : Haemorrhage, Melanoma, Distomatosis (liver flukes)

(B) Myocardial lipofuscinosis (Brown atrophy of the heart, Xanthosis)

Xanthosis ("Wear-and-Tear") pigment is a brown pigmentation of skeletal and heart muscles of cattle (Fig. 20). The condition is seen in old animals such as "cull dairy cows" and in some chronic wasting diseases. It is prevalent in Ayrshire cows and approximately 28 % of normal Ayrshire cows have this pigment

in skeletal and heart muscles. Xanthosis is not dependent on the age of animals in this breed.

Fig. 19: Melanin deposits in the sheep viscera.

Fig. 20: Myocardial lipofuscinosis.

(C) Congenital porphyria (osteohemochromatosis, pink tooth)

Porphyria is the accumulation of plant or endogenous porphyrins in the blood resulting in tissue pigmentation and photosensitization. This is a hereditary disease and is observed in cattle, swine and sheep. In porphyric cattle, exposure to light will initiate the development of photodynamic dermatitis. In swine, photodynamic dermatitis does not occur.

The disease is also known as osteohemochromatosis, due to a reddish brown bone pigmentation (Fig. 21), and "pink tooth" because of a brownish-pink discoloration of teeth.

Judgement : Carcass showing extensive xanthosis is *condemned*. If the condition is localized, *only the affected* organ or part of the carcass needs to be condemned. Head and bones of a carcass affected with osteohemochromatosis are condemned. The bones are "boned out" and remaining muscles are approved. If the condition is generalized the carcass is condemned.

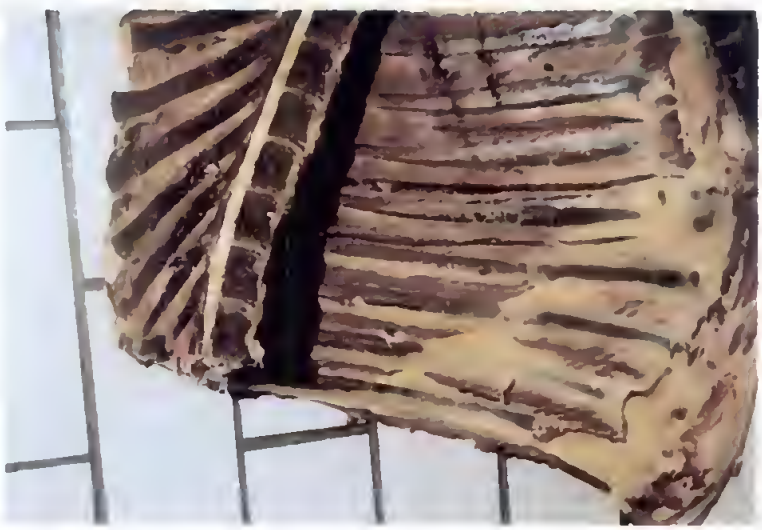


Fig. 21: Osteohemochromatosis showing brown pigmentation of ribs and vertebrae in a 6 months old calf.

(D) Icterus(Jaundice)

Icterus is the result of an abnormal accumulation of bile pigment, bilirubin, or of haemoglobin in the blood. Yellow pigmentation is observed in the skin, internal organs (Fig. 22, 23), sclerae (the white of the eye), tendons, cartilage, arteries, joint surfaces etc. Icterus is a clinical sign of a faulty liver or bile duct malfunction, but it may be also caused by diseases in which the liver is not impaired. Jaundice is divided into three main categories (Fig. 24).

1. Prehepatic jaundice (haemolytic icterus)
2. Hepatic jaundice (toxic icterus)
3. Posthepatic jaundice (obstructive icterus)

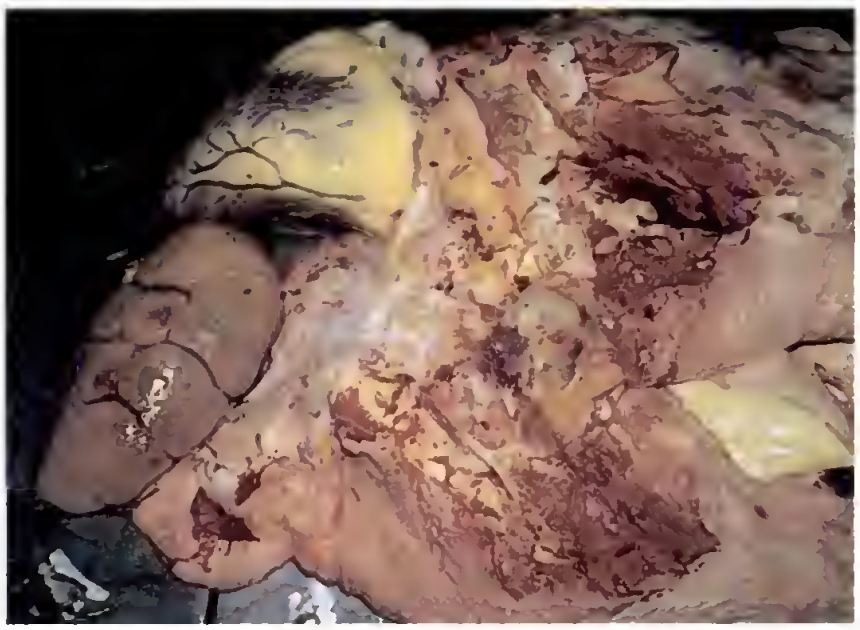


Fig. 22: Jaundice of an aged cow caused by liver disease. Note yellow discoloration of body fat, lungs, heart and kidneys.

Fig. 23: Yellow discoloration of pig viscera and carcass caused by cirrhosis of the liver.

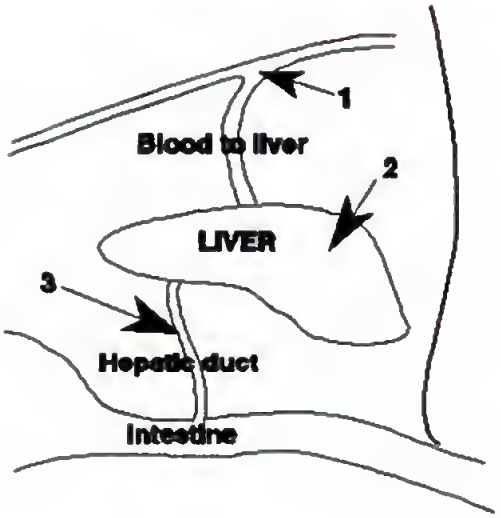


Fig. 24:
Classification of jaundice
1. Pre-hepatic;
2. Hepatic;
3. Post-hepatic

Prehepatic jaundice occurs following excessive destruction of red blood cells. Tick-borne diseases such as Babesia ovis and Anaplasmosis cause this type of icterus, which is one of the main causes of carcass condemnation in Southern Africa due to

prevalence of these parasites. Overproduced blood pigment, which cannot be metabolized in the liver, builds up in the blood (haemoglobinemia). It is excreted by the kidneys into the urine (haemoglobinuria). Normal urine colour changes and becomes bright red to dark red.

Hepatic jaundice occurs due to direct damage to liver cells as seen in liver cirrhosis (Fig. 23), systemic infections, and in chemical and plant poisoning. In sheep, jaundice may have been caused by phytogenic chronic copper poisoning.

Liver function is impaired and the liver is unable to secrete bile pigments. Obstructive jaundice occurs when the drainage of the bile pigment bilirubin is blocked from entry into the intestine. This usually occurs due to the obstruction of the hepatic ducts by a tumour, by parasites such as flukes or by gall stones. Obstruction may also occur due to an inflammation of the bile ducts. In hogs, mature ascarides may occlude the bile ducts.

Judgement : Animals suspected to have icterus should be treated as "suspects" on antemortem examination. On postmortem examination, the carcass and viscera with haemolytic, toxic icterus and obstructive icterus are *condemned*. Less severe cases are kept in the chiller for 24 hours. Upon re-examination, the carcass may be *approved or condemned* depending on the absence or presence of pigment in the tissue. If the obstructive icterus disappears after 24 hours, the carcass and viscera can be *passed* for human food.

A simple laboratory test will help to make an objective test for bile pigment icterus. Two drops of serum are mixed on a white tile with two drops of Fouchets agent.[2] A blue/green precipitate is

[2] Fouchets Reagent Trichloroacetic acid 25 gm
$FeCl_3$ (10 % solution) 10 ml
Distilled water 100 ml

positive for bile icterus.

Differential diagnosis : Yellow fat in animals with heavy corn rations, nutritional panniculitis (yellow fat disease, steatitis) and yellow fat seen in extensive bruises. In yellow fat disease, the fat has a rancid odour and flavour upon cooking.

To differentiate icterus from the normal colour of fat of certain breeds, the sclera, intima of the blood vessels, bone cartilage, liver, connective tissue and renal pelvis should be examined. If yellow discoloration is not noted in these tissues, icterus is not present.

Icterus should not be confused with yellow fat disease in hogs fed predominantly on fish by-products or by the yellowish appearance of tissue caused by breed characteristics or nutritional factors.

Haemorrhage and Haematoma

Haemorrhage is seen at slaughter in various organs, mucous and serous membranes, skin, subcutaneous tissue and muscles. It may be caused by trauma, acute infectious diseases or septicemia.

In pigs muscles haemorrhage is frequently associated with fractures (Fig. 25). Petechial haemorrhage is noted as tiny foci 1 - 2 mm in diameter. Ecchymotic haemorrhage (Fig. 26) is larger being up to 2 - 3 cm in size. Paint brush haemorrhage includes extensive streaking with haemorrhage. Haemorrhage is also associated with vitamin C deficiencies, a sudden increase in blood pressure with weakened blood vessels, and improper electric current stunning in pigs and sheep. Lengthy transportation, exposure to stress before slaughter, hot weather and excitement are some of the other factors which contribute to muscle haemorrhage.

Fig. 25: Fractured bone and muscle haemorrhage in a market hog.

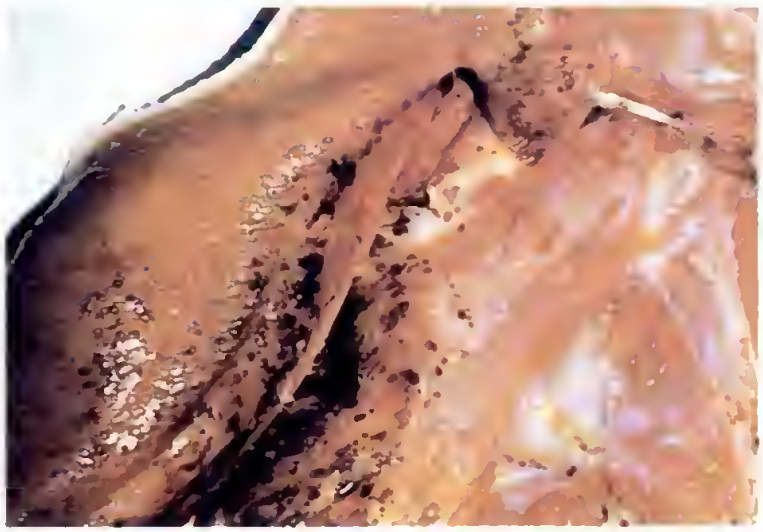


Fig. 26: Ecchymotic haemorrhage in the tongue muscle of a cow.

In haemorrhage caused by improper stunning, there may be a delay between stunning and sticking of the animal. The electrical current used in stunning causes cardiac muscle stimulation and vasoconstriction of blood vessels. This might induce a rapid rise in blood pressure leading to haemorrhages in the organs and muscle (so called "blood splashing").

The stunning of animals by a mechanical blow to the head is still practised with sheep and is a significant cause of haemorrhage in organs particularly the lungs and heart. The blow to the head will initiate a rise in blood pressure. The normal arterial blood pressure in sheep is 120 - 145 mm/Hg. This may rise to 260 mm/Hg or over in a stunned animal. The heart rate will be increased. Immediate bleeding with the fast blood flow from the cut vessels could prevent this type of haemorrhage in sheep.

Agonal haemorrhage (due to rupture of capillaries) is caused by laboured breathing and contraction of musculature during violent death.

A lump formed from a blood clot in tissues or organs is called a haematoma. Haematoma varies in size and may be over one meter in diameter (Fig. 27). They are associated with trauma or a clotting defect. Haematoma of the spleen (Fig. 28) may be associated by head butting by horned animals.

Judgement : A carcass is *approved* if the haemorrhage is minor in extent and is due to physical causes. The affected tissue is *condemned*. A carcass affected with extensive haemorrhage where salvaging is impractical, or a haemorrhagic carcass associated with septicemia is *condemned*.

Differential diagnosis : Haemorrhage resulting from blackleg, and sweet clover poisoning.

Fig. 27: Haematoma in the abdominal wall of an ewe.

Fig. 28: Haematoma of the bovine spleen.

Bruises

Bruises are frequently found on antemortem and post-mortem examination in food producing animals and poultry. In cattle bruises caused by transportation or handling are commonly found in the hip, chest and shoulder areas; in pigs within the ham and in sheep in the hind leg. Bruises and haemorrhage in the hip joint are caused by rough handling of animals during shackling. Bruises in poultry can be localized or generalized and are frequently associated with bone fractures or ruptured ligament tendons.

Judgement : Bruised animals should be treated *as suspects* on ante mortem examination. On postmortem examination, carcasses affected with local bruising are *approved* after being trimmed. Carcasses affected with bruises or injuries associated with inflammatory lesions are also *approved* if tissue reaction *does not extend beyond the regional lymph nodes*. The affected area should be *condemned*. When bruises or injuries are associated with systemic change and the wholesomeness of the musculature is lost, the carcass will be *condemned*.

On postmortem examination of bird carcasses affected with bruises and fractures, the following judgement should be observed: (a) the fractures associated with bruises are *removed* and affected tissue is *condemned*, (b) in compound fractures with damaged skin, the fractured site and surrounding tissue are condemned; (c) in simple fractures without bruises and damaged skin, the affected portion *may be approved* for mechanical and manual boning operations. If the lower part of bone is fractured, the bone may be *removed* by cutting above the fracture. A carcass affected with extensive bruises is *condemned* on postmortem examination (Fig. 29). A slightly or moderately bruised carcass is *approved* if no systemic changes are present. Affected tissues are *condemned*.

Fig. 29: Extensive bruises of a beef carcass.

Abscess

An abscess is a localized collection of pus separated from the surrounding tissue by a fibrous capsule.

The most common bacteria in liver abscesses include Actinomyces (Corynebacterium) pyogenes, Streptococcus spp. and Staphylococcus spp. In the lungs the most common bacteria are Pasteurella spp. and Actinomyces pyogenes. Fusobacterium (Sphaerophorus) necrophorum causes liver abscesses (Fig. 30) as a complication of rumen inflammation (rumenitis) in adult cattle. This condition is common in feedlots where cattle are fed a high grain diet which produces acidity in the rumen and ulcerative rumenitis. The rumen lesion is invaded by F. necrophorum which pass further via the veins to the liver and stimulate abscess formation.

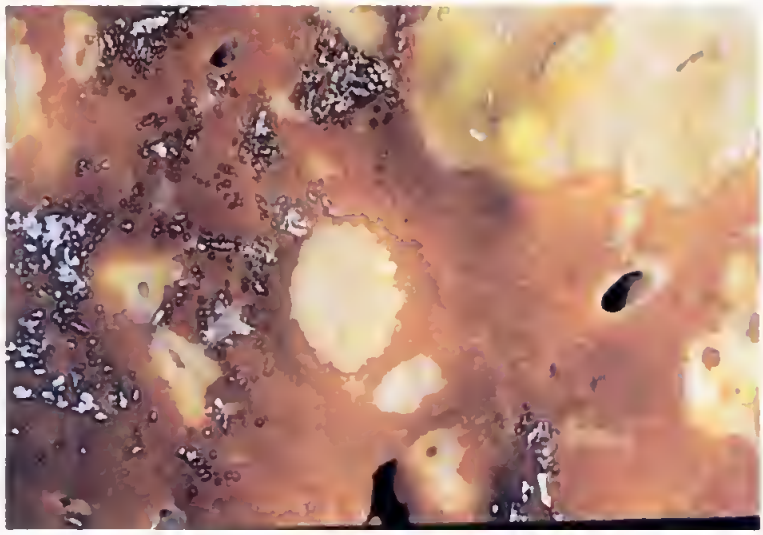


Fig. 30: Liver abscesses caused by Fusobacterium necrophorum.

Judgement : The judgement of animals and carcasses affected with abscesses depends on findings of primary or secondary abscesses in the animal. The portal of entry of pyogenic organisms into the system is also of importance. The primary abscess is usually situated in tissue which has contact with the digestive tract, respiratory tract, subcutaneous tissue, liver etc. The secondary abscess is found in tissue where contact with these body systems and organs is via the blood stream. The brain, bone marrow, spinal cord, renal cortex, ovary and spleen (Fig. 31) may be affected with secondary abscesses. In judgement of the carcass, the inflammation of the renal medulla and contact infection in the spleen and ovaries must be ruled out. A single huge abscess found in one of the sites of secondary abscesses may cause the condemnation of a carcass if toxaemia is present. In pigs an abscess is frequently observed in the jaw and in the spine. Spinal abscesses in pigs are commonly caused by tail biting (Fig. 32). The bacterial agent from the tail penetrating the spinal canal could be arrested in the lumbo-sacral and cervical spinal enlargements, initiating an abscess formation.

Inspectors should differentiate the abscesses in the active and growing state from the older calcified or healed abscesses. In domestic animals, the primary sites of purulent infections are post-partum uterus, umbilicus or reticulum in "hardware disease". Secondary abscesses are frequently observed in distant organs. Small multiple abscesses may develop in the liver of calves as a result of infection of the umbilicus ("sawdust liver", Fig. 33). Carcasses with such condition should be condemned.

The animals affected with abscesses spread through the blood stream(pyemia) are *condemned* on antemortem if the findings of abscesses are over most areas of the body and systemic involvement is evident as shown in elevated temperature and cachexia.

On postmortem examination, the carcasses are *condemned* for abscesses, if the abscesses resulted from entry of pyogenic organisms into the blood stream and into the abdominal organs, spine or musculature. An abscess in the lungs may require *condemnation* of the lungs and an *passing* the carcass if no other lesions are noted. Liver abscesses associated with umbilical infection require *condemnation* of the carcass. If no other infection is present the abscess is *trimmed off* and the liver may be utilized for human or animal food depending on the regulations of the respective country. Multiple abscesses in the liver require condemnation of the organ.

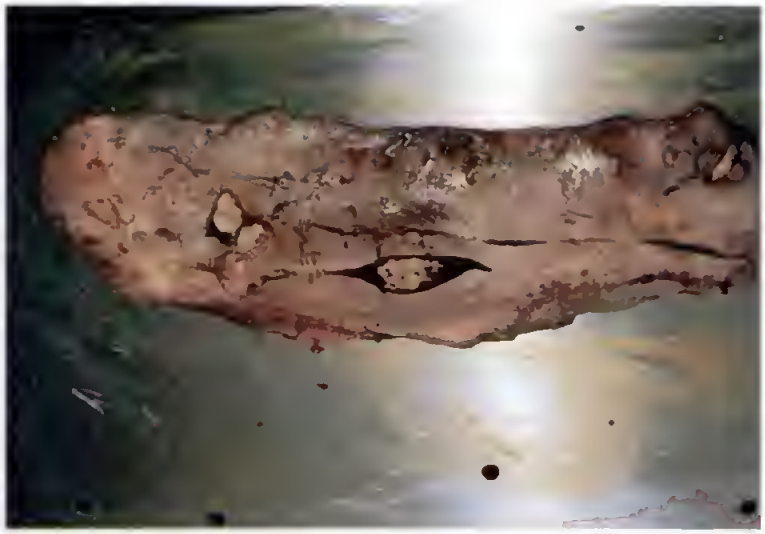


Fig. 31: Secondary abscesses in the spleen of an aged cow.

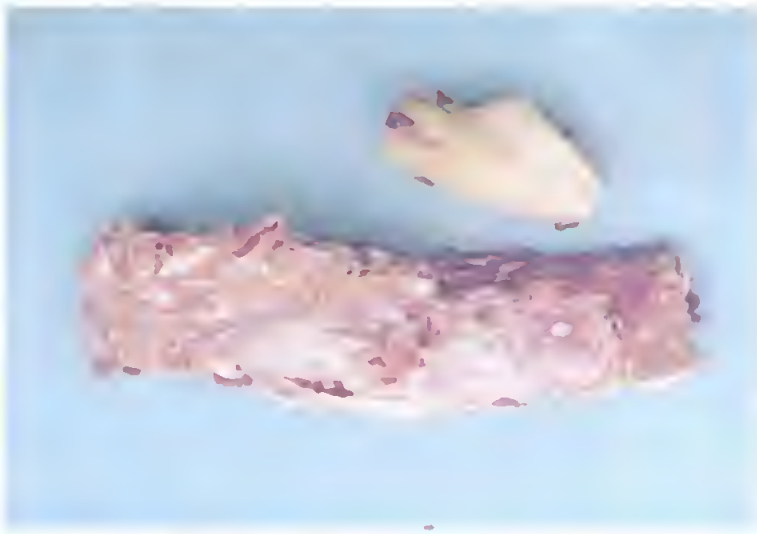


Fig. 32: Tail necrosis caused by biting and secondary spine abscesses.

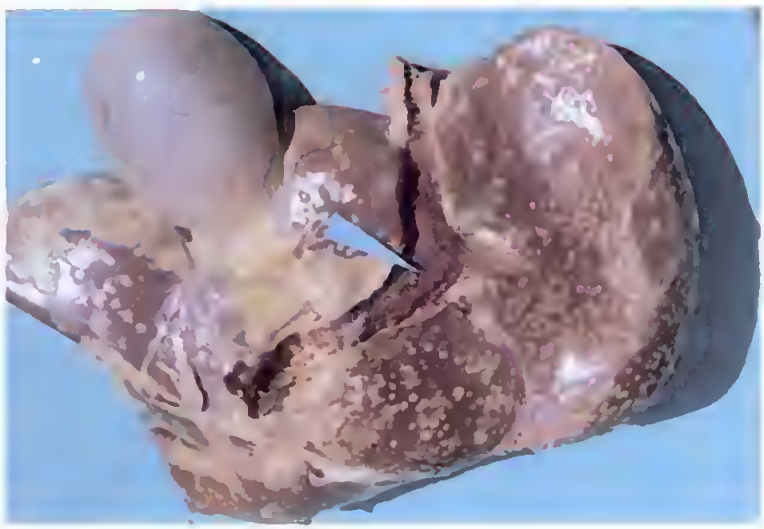


Fig. 33: Multiple abscesses in the calf liver as a result of an umbilical infection; carcass with such condition should be condemned.

Emaciation

Emaciation is a common condition of food animals and is characterized by a loss of fat and flesh following the loss of appetite, starvation and cachexia. It is associated with gradual diminution in the size of organs and muscular tissue as well as edema in many cases. The organs and muscular tissue appears thinner, moist and glossy. Cachexia is a clinical term for a chronic debilitating condition or general physical wasting caused by chronic disease.

Emaciation may be associated with chronic diseases and parasitic conditions such as round worms in pigs and fascioliasis in cattle and sheep, swine erysipelas, neoplasms, tuberculosis, John's disease, caseous lymphadenitis, and poor teeth and lack of nutrition. Emaciation is a postmortem descriptive term which should be differentiated from thinness.

Antemortem findings :

1. Wrinkled, dry leathery skin (Fig. 34)
2. Rough hair coat
3. Prominent bones and sunken eyes

Postmortem findings :

1. Serous atrophy of fat in the carcass and organs especially the pericardial and renal fat (Fig. 35).
2. The fat is watery. Tranluscent or jelly-like and hangs from the intervertebral spaces (Fig. 36).
3. Edema and anaemia may develop due to starvation and malnutrition due to parasite infestations.

Judgement : Animals affected with emaciation should be treated as *"suspects"* on antemortem inspection. On postmortem examination it is important to assess and differentiate emaciation from leanness. In case of doubt, the carcass may be held in the

refrigerated room and the general setting of the carcass should be examined the following day. If the body cavities are relatively dry, edema of muscle tissue is not present and fat is of an acceptable consistency i.e. has "set", the carcass may be *passed* for food.

Well nourished carcasses with serous atrophy of the heart and kidneys and mere leanness may also be *fit* for human consumption. A carcass with any amount of normal fat may be *approved* if everything else appears normal. The carcasses from animals being in transport for a long period of time may show extensive serous atrophy of fat (mucoid degeneration of fat tissue) without any changes in organs and muscles. If after being in the cooler for 24-48 hours, the fat resumes its normal consistency, the carcass is *approved*. Otherwise, the carcass is *condemned*.

The carcass and viscera must be *condemned* if emaciation is due to chronic infectious disease. An objective judgement of emaciation with edema may be made using a 47 % ethanol/methanol in water solution. A clear, pea-sized piece of bone marrow, taken from the distal radius, is put carefully into the solution. If it sinks, the marrow which reflect the water content of the carcass as a whole, has approximately 45 % water content. The carcass should be *condemned*.

Differential diagnosis : Thinness-leanness, edema and uraemia.

Leanness (Poorness) is often observed in range bulls on poor quality pasture, high milking cows and young growing animals which have had protein deficient diet. The animals are physiologically normal and the reduced fat deposits of the animal carcass are normal in colour and consistency. The reduced muscle tissue is firm and of a normal consistency. The muscle colour is darker than normal, and fat tissue may still be present in the orbit of the eye.

Fig. 34: Emaciated cow showing marked reduction of muscle mass.

Fig. 35: Serous atrophy of renal fat. Note petechial haemorrhages, seen frequently in septicemic diseases.

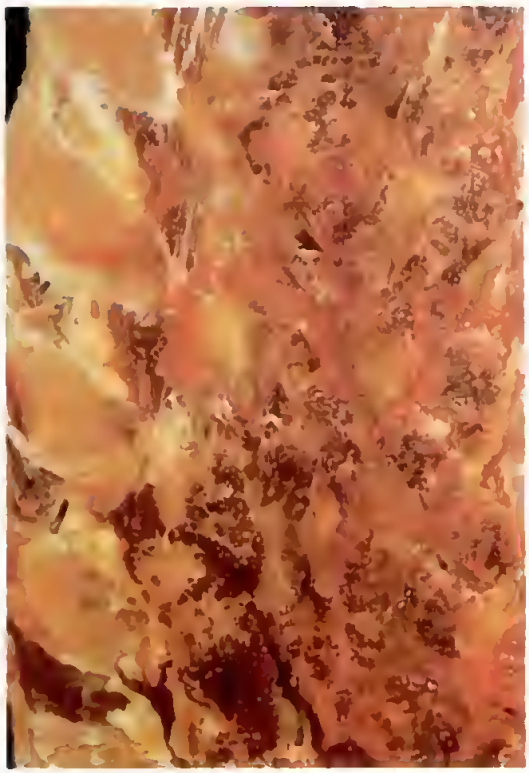


Fig. 36: Hanging gelatinous fat between the spinal processes.

Edema

Edema is the accumulation of excess fluid in the intercellular (interstitial) tissue compartments, including body cavities.

There are two types of edema:

1. Inflammatory edema (exudate)
2. Non-inflammatory (transudate)

Inflammatory edema shows yellow, white or greenish clear or cloudy fluid in the area of inflammation. *Non-inflammatory edema* is an accumulation of fluid in subcutaneous tissue, submucosae, lungs and brain.

Localized edema is noted after:

a. The swelling of a leg of a cow in prolonged decubitus. This

swelling is caused by obstruction of the venous outflow

b. Interference with the lymph circulation of an organ or area by proliferation of tumours in or around bile ducts.

c. Inflammation or an allergic reaction

Systemic or generalized edema may occur secondary to congestive heart failure or is caused by low protein levels in the blood. The latter may be associated with:

- severe malnutrition
- severe amyloidosis of the kidney
- gastrointestinal parasitic infestation
- chronic liver disease
- damage to the vascular endothelium by toxins and infectious agents

Anasarca is a form of edema of the subcutaneous tissues. Ascites is an accumulation of fluid in the peritoneal cavity. Hydrothorax is an accumulation of fluid in the pleural cavity. Hydrothorax may accompany traumatic pericarditis, ascites, cirrhosis of the liver and round worm infestation in sheep. Anasarca may be caused by toxaemic infection.

Antemortem findings :

1. Depressed and drowsy
2. Swelling of the mandible, dewlap, legs, shoulder, brisket and abdomen (Fig. 37)
3. Edematous tissue is cool upon touch and is of a firm, doughy consistency.

Postmortem findings :

1. Wet, sloppy musculature which pits on pressure
2. Accumulation of clear or faint yellow fluid in the thorax, abdomen and subcutaneous tissue

Judgement : Animals affected with generalized edema may be *condemned* on antemortem inspection. In less severe non-generalized cases, animals are treated as *"suspects"*. When making a judgement of a carcass affected with edema, it is important to know the underlying cause of the edema and also to know the significance of all other lesions found in the carcass.

The carcass may be *totally or partially condemned* depending on the extent and cause of the condition. The presence of localized edema necessitates *removal* of the affected area. The carcass is then *approved*. Edema associated with diseased conditions such as traumatic pericarditis, malignant neoplasm or septicemia requires *condemnation* of the carcass because of the primary condition. Edema observed in the mesentery is commonly related to circulation interference in the caudal vena cava due to liver abscess or chronic liver disease. Such a carcass may be held in the cooler for re-examination. Dry serous membranes of the abdominal and thoracic walls and a carcass appearing normal after re-examination can be *passed for* human consumption. Carcasses which have been condemned for edema associated with malnutrition only may be *salvaged for animal food* (except in case of edema associated with septicemia).

Differential diagnosis : Pericarditis, peritonitis, pleuritis, renal amyloidosis, liver disease, grain overload and vagal indigestion, high altitude disease, uraemia and absorption of a large bruised area.

Fig. 37: Abdominal edema caused by liver disease.

Emphysema

Emphysema in animals is associated with some disease conditions and is caused by an obstruction to the outflow of a air or by extensive gasping respiration during slaughter procedures.

All species may be affected with alveolar emphysema. However interstitial emphysema (Fig. 38) occurs mostly in cattle. In the latter, the lack of collateral ventilation forces the rupture of alveoli and the migration of air into the interstitium. The lobules of the lungs become separated by the distended interstitial tissue and marked lobulation of lungs is observed.

Alveolar emphysema appears as small air bubbles due to air trapped in dilated alveoli. Large accumulations of air, a few centimetres in diameter, are called "bullae or bullous emphysema".

Postmortem findings : Postmortem findings of the emphysematous lungs include a pale, enlarged greyish-yellow,

pearl like shiny lesion. Upon palpation, the affected area feels puffy and crepitant.

Two diseases of food animals associated with emphysema are *chronic obstructive pulmonary disease* (COPD) in horses, and *interstitial pneumonia* in cattle. COPD is also called heaves and frequently described under chronic bronchitis or bronchiolitis in horses. Interstitial pneumonia in cattle is also described under fog fever or acute chronic pulmonary edema and emphysema.

Judgement : Affected lungs are *condemned.*

Fig. 38: Interstitial emphysema in the cow's lungs.

Tumours or neoplasms

A tumour is an abnormal mass of tissue which grows without control and uncoordinated with the tissue or organs of origin or those nearby. Its presence if often cumbersome to the tissue or organ it arose either by pressure or by replacement of normal functional tissue. Tumour cells resemble healthy cells however serve no useful purpose. The term *tumour* in current medical lexicon is presently limited to neoplastic growths.

Tumours are usually divided according to tissue of origin i.e. epithelial, mesenchymal (connective tissue), haemopoietic, nervous etc. Tumour behavioral classification include their mode of growth and the degree of invasiveness. Slow growing non invasive circumscribed tumours are considered *benign* and fast growing, infiltrative and frequently metastatic are *malignant* tumours. The spread of neoplasm is by direct expansion and infiltration, via lymphatics and blood circulation and by implantation. *Carcinoma* are tumours of the epithelial tissue. They are usually spread via the lymphatic system. *Sarcoma* are connective tissue tumours, commonly spread via haematogenous route. Implantation to surrounding parietal cavities is observed in ovarian carcinoma. The spread of malignant tumours via lymphatics or haematogenous spread to another area not directly connected with the original site is called metastasia.

Some of the common tumours found during beef inspection are *squamous cell carcinoma, lymphosarcoma, pheochromocytoma, mesothelioma* etc.

Fig. 39: Squamous cell carcinoma affecting cow's eye and parotic lymph node. In this case, tumour lesions were also observed in the lungs. Carcass was condemned.

Judgement : Carcass affected with metastatic neoplasms is *condemned*. Multiple benign tumours in different organs also require *condemnation* of carcass. Carcass affected with circumscribed benign tumours is *approved*.

Calcification

Calcification is the deposition of calcium salts in dead and degenerating tissue. It may be regarded as a body reaction to immobilize some foreign agents. It may occur in any tissue or organ. In dairy cows, calcification is noted in the heart (*endocardium*) and is caused by excessive dietary supplementation with Vitamin D. In cattle mineralization of the *aorta* and *brachiocephalic trunk* (Fig. 40) is sometimes seen. Calcification is also seen in *parasitic infections* (Fig. 41) and in many *chronic infections* such as tuberculosis, botryomycosis etc. The presternal pressure necrosis of fat (*putty brisket*) seen in cattle and rarely in sheep may also eventually mineralize. Inflammatory metaplasia leading to ossification is an incidental finding during post-mortem examination of food animals. It is most commonly found in *peritoneal scars* of hogs.

If calcium particles are removed from the surrounding tissue, they appear white or grey, irregularly rounded and frequently honeycombed. Calcification is detected on postmortem examination by a gritty sound upon incision with a knife.

Judgement : Carcass and viscera affected with presternal calcification are *approved*. Affected brisket is *condemned*. Calcified parasitic organs and heart in dairy cows are also *condemned*.

Fig. 40: Calcification of the aorta and brachiocephalic trunk in a young heifer.

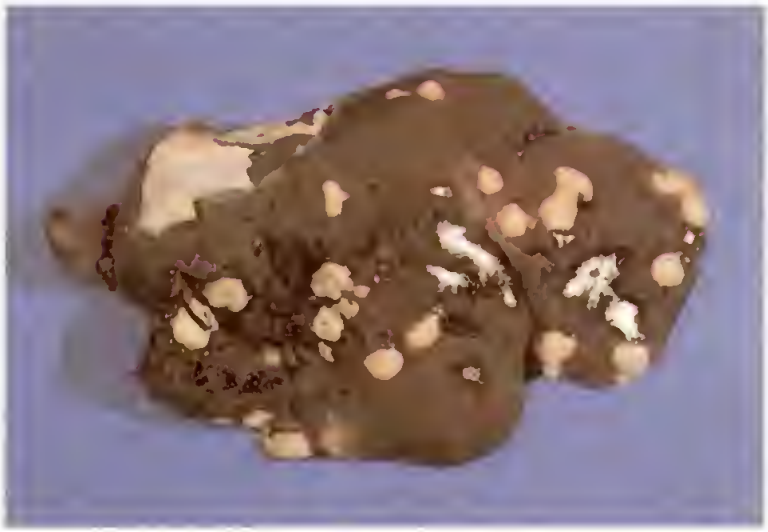


Fig. 41: Parasitic lesions undergoing mineralization in a sheep liver.

Degeneration

Degeneration implies the change of tissue to a lower or less functionally active form or deterioration (impairment) of an organ or cell due to changes in its size. If chemical change of the tissue occur this is regarded as a true degeneration. Cloudy swelling

(parenchymatous degeneration, albuminous degeneration or granular degeneration, acute cellular swelling) in the cell is a response to cell insults including trauma, anoxia, immune mechanisms, toxins, viral, and bacterial agents. In *cloudy swelling*, cell proteinaceous substances become cloudy and the cell increases in size. It is observed in the *heart, kidneys, liver, glands* and *muscles*.

Cloudy swelling is often associated with *fatty degeneration*. Affected organs are pale, lustrous and softer than normal, slightly enlarged and have the appearance of having been boiled (Fig. 42). In slight insults, the animal may recover and in severe cases cloudy swelling is succeeded by fatty degeneration.

Fatty infiltration is an accumulation of fat in the *heart, liver, kidneys, pancreas*, etc. The liver is yellow, soft in consistency, has round edges, dimples on pressure, is enlarged and has a greasy texture on cut surfaces. Fatty infiltration may disappear from the tissues if the causative agent is removed. The extensive accumulation of fat in the liver is caused by an increased dietary intake of fat, increased mobilization of fat during lactation or starvation. It is also seen in healthy animals slaughtered shortly after parturition and often accompanies advanced pregnancy in cows and ewes.

Fatty degeneration is an irreversible process and occurs when fat accumulates in the damaged cell. The liver capsule is dull and has a turgid appearance. This condition is associated with acute febrile and toxic conditions and with chemical poisoning by arsenic, phosphorus, chloroform etc. The liver and kidneys affected are a pale, clay-red colour and greasy on touch. They have a patchy or spotted appearance.

Judgement : Organs and muscles affected with cloudy swelling are *condemned*. Detailed examination of the carcass is necessary

since systemic changes are usually present and the carcass is therefore *condemned*. A liver affected with fatty infiltration is *approved*.

Fig. 42: Degeneration. Cloudy swelling and associated fatty change of the pig liver.

Telangiectasis ("Plum pudding", Fig. 43)

This liver condition is found in cattle, sheep and horses. It is more frequent in older cows. The liver lesions are bluish black and irregular with depressed surfaces and dilated blood filled hepatic sinusoids. A cause of hepatic telangiectasis in cattle is thought to be local ischemia.

Judgement : Slightly affected liver is *approved* after appropriate trimmings. Extensively affected liver requires *condemnation*. Condemned material can be used for animal food.

Fig. 43: Telangiectasis. Bovine liver affected with telangiectasis.

Abnormal odours

Abnormal odours may result from the ingestion of certain *feedstuff, drugs, various pathological conditions, absorption of odours* from strong smelling substances and *sexual odour* from some male animals. Pig carcasses may have a fishy odour if the pig was consuming excessive fish meal in the diet or was fed cod fish oil. Drugs which may cause absorption of odours include turpentine, linseed oil, carbolic acid, chloroform, ether, aromatic spirits of ammonia etc.

In cows affected with ketosis, the sweetish odour of acetone may be present in the muscles. If treatment was not successful in dairy cows affected with milk fever, the odour of acetone may be noted in the connective tissue, kidney fat and musculature. The flesh of bloated and constipated animals may give off a faecal odour. If the meat is kept in a room which was recently painted, the odour may pass on to the carcass. The odour is most noted in a carcass right

after slaughter.

Judgement : The carcass having fish meal odour has *inferior* meat. Viscera and organs are also inferior. Generalized drug treatment requires *condemnation* of the carcass. If local treatment and withholding periods are observed, the carcass and viscera are *approved*. Sexual odour in a carcass can have a *limited* distribution according to the consumers taste. Extremely strong sexual odour requires *condemnation* of the carcass.

A carcass which gives off a pronounced odour of medicinal, chemical or other foreign substances shall be *condemned*. If the odour can be removed by trimming or chilling, the carcass *may be passed* for human food after the removal of affected parts or dissipation of the condition.

Carcasses affected with sexual odour should be held in the cooler and *re-tested* periodically. If the odour disappears the carcass is *approved*. If the sexual odour is present after 48 hours, the carcass shall be *condemned*. Young boars and ridglings are treated as suspects and held pending a heat test.

If abnormal odour is suspected the smell will be enhanced by placing a piece of muscle or tissue in cold water and bringing to the boil.

Immaturity

Immaturity occurs mainly in calves. In many countries, the slaughter of calves younger than two weeks of age is prohibited. The muscle of immature animals is moist, pale, flabby and poorly developed. It is low in protein, high in water content and contains a high proportion of bone. Immature animals should not be

slaughtered for human consumption.

Antemortem and postmortem findings :

1. Presence of the umbilical cord
2. Bluish and not completely retracted gums
3. Greyish muscles are flabby, tear easily and are not well developed
4. Dark red kidney and edematous kidney capsule

Judgement : Carcass and offal of immature animals are *condemned.*

Remarks : A presence or non presence of fat around the kidneys ("caul fat") should not be used as a guide for judgement of immature animals.

Plant poisoning

In developing countries, slaughter animals, particularly cattle are often trekked some hundreds of kilometres on the hoof to the abattoirs. During this journey, animals may suffer from various plant poisoning. In addition cattle living in areas where pasture has poisonous plants may suffer from the effects of chronic plant poisoning. Different body systems may be affected and various lesions may be seen at meat inspection.

Clinical signs and gross lesions observed in animals that have ingested certain poisonous plants: Tulip (*slangkop*) causes *diarrhoea, bloated abdomen* and *heart failure.* Lantana camara causes *photosensitization.* Senecio causes *necrosis* and *cirrhosis of liver.* Crololaria causes *laminitis.* Dicapetalum cymosum causes *heart failure* and *sudden death.*

Judgement : Judgement of the animal or animal carcass will *depend on the clinical signs* and the extent and severity of the lesions.

Chemical poisoning

Dipping of cattle in acaricide on a regular basis is practised in many parts in order to control thick borne diseases. Chemicals used for this purpose include *arsenic, chlorinated hydrocarbons* and *organophosphates*. Dipping may lead to clinical cases of poisoning, which may be manifested with the following clinical signs: *nervous system disturbances, acute abdominal pain, diarrhoea and skin lesions*. Gross lesions may include *gastro-enteritis, fatty degeneration of the liver* and *inadequate bleeding*.

Judgement : The carcass, offal and intestine should be *condemned if clinical signs* of poisoning are *associated with postmortem lesions*.

Spear grass penetration of sheep

Grassland in many parts of Africa contains scattered grasses with spear-like seeds. These seeds may penetrate through the wool and skin to the subcutis, and further through to the abdominal wall into the abdominal cavity.

Antemortem and postmortem findings :

1. Spear-like seeds in the wool and skin
2. Spear-like seeds in the connective tissue, fat and musculature

(Fig. 44)

3. Acute inflammation of the affected tissue
4. Abscessation
5. Spear-like seeds in the abdominal cavity causing low grade peritonitis

Judgement : If an acute generalized inflammation is associated with haemorrhages and abscesses, the carcass should be *condemned*, otherwise the carcass is *approved*.

Fig. 44: Spear grass penetration of sheep. Numerous spear like seeds in the sheep carcass.

CHAPTER 3

SPECIFIC DISEASES OF CATTLE

Diseases caused by viruses

Foot and mouth disease (FMD, Aphthous fever)

FMD is an acute viral and extremely contagious disease of cloven footed animals such as cattle, sheep, goats, pigs and antelope. It is manifested by vesicles and erosions in the muzzle, nares, mouth, feet, teats, udder and pillar of the rumen. There are three main strains of viruses causing FMD, namely A, O and C. Three additional strains, SAT 1, SAT 2 and SAT 3 have been isolated from Africa and a further strain ASIA-1 from Asia and the Far East.

Transmission : Direct and indirect contact with infected animals and their secretions including saliva, blood, urine, faeces, milk and semen, aerosol droplet dispersion, infected animal by-products, swill containing scraps of meat or other animal tissue and fomites and vaccines.

Antemortem findings :

Before vesicle formation:

1. Incubation is 1 - 5 days or longer
2. Morbidity: Nearly 100 %
3. Mortality: variable depending on the strain of virus and its virulence and susceptibility of host; 50 % in young animals, 5 % in adults
4. Fever up to 41.7°C
5. Dullness
6. Lack of appetite

7. Drastic drop in milk production.
8. Uneasiness and muscle tremors

Vesicle formation:

9. Smacking and quivering of lips
10. Extensive salivation (Fig. 45) and drooling
11. Shaking of feet and lameness

The vesicles and later erosions are commonly found on the muzzle, tongue (Fig. 46), oral cavity, teat and on the skin between and above the hoofs of the feet. In more chronic cases in cattle the hoof become loose and the animal may walk with characteristic "clicking" sound (Slippering).

Some strains of FMD, particularly in swine, sheep and goats cause erosions instead of vesicles.

Postmortem findings :

1. Necrosis of heart muscle(tiger heart), usually only in young acutely infected animals.

2. Ulcerative lesions on tongue, palate, gums, pillars of the rumen and feet.

Judgement : In countries or in zones within a country free or nearly free of FMD diseased or suspect animals are *prohibited to be admitted in an abattoir* or slaughtered. If FMD is suspected on postmortem examination the carcass and viscera are *condemned* and appropriate action recommended by the regulatory authorities of the country must be taken. In countries where this disease is present, the judgement should be *in accordance with the current animal health requirements*, and consisted with effective public health protection. Particular attention should be paid to secondary

bacterial infections and general findings. Sanitary measures should be taken to comply with national animal health policy.

Remarks : Latent infections with Salmonella organisms were reported in animals affected with FMD.

Differential diagnosis in bovine and ovine species : Vesicular stomatitis, allergic stomatitis, feedlot glossitis, photosensitization, bluetongue, rinderpest, infectious bovine rhinotracheitis, malignant catarrhal fever, bovine papular stomatitis, bovine viral diarrhoea, pseudocowpox, ovine pox, contagious ecthyma, footrot, mycotoxicosis and increased salt in concentrate.

Discussion : In order to prevent the spread of the virus in the abattoir, the equipment and room should be disinfected with 2 % NaOH (caustic soda). In some countries sodium carbonate (Na_2CO_3) is used. The vehicle conveying diseased animals should also be disinfected and abattoir personnel leaving the abattoir should pass through a footbath with 1 % solution of NaOH.

The virus of FMD can survive in meat and meat products for a considerable length of time. Outside the pH range of 6 - 9, viral infectivity is destroyed. A bovine carcass matured at above +2°C produces a drop in the pH of muscle tissue to between 5.3 - 5.7 within 24 hours of slaughter. This is caused by the formation of sarcolactic acid. Quick freezing of the meat arrests acid production and consequently the virus remains infective for about 6 months. In salted meat at 4°C, the virus is still infective in bone marrow and lymph nodes for 6 months. In blood clots in large vessels of cattle and swine, the virus is infective for 2 months. The virus is inactivated by ultraviolet rays, acetic acid, 2 % lye and ethylene oxide. At high temperatures, the virus is only active for a short period. 2 % NaOH solution inactivates the virus in 1 - 2 minutes. In dry refuse in stalls, the virus remains infective for 14 days, 3 days on soil surfaces in summer compared to 39 days in fall. It is

also infective for 39 days in urine and for 20 weeks on hay dried at 22°C. The virus can be destroyed with 0.5 % citric or lactic acid, by cooking meat to an internal temperature of 69°C and by pasteurization processes of milk.

Fig. 45: Excessive salivation in a cow affected with FMD.

Fig. 46: FMD. Extensive areas of eroded epithelium on a bovine tongue.

Rinderpest (RP)

Rinderpest is an acute, highly contagious, fatal viral disease of *cattle, buffalo* and *wild ruminants* manifested by inflammation, haemorrhage, erosions of the digestive tract, wasting and often bloody diarrhoea. Some swine species are also susceptible. Man is not susceptible to RP virus.

Transmission : Direct contact with infected animals or their

excretions and secretions and fomites. The virus appears in the blood and in secretions before the onset of clinical signs and this may cause infection in abattoirs and stockyards.

Antemortem findings :

1. Incubation: 3 - 10 days or longer
2. Morbidity: Up to 100 % in a susceptible herd
3. Mortality: 50 % and may reach 90 - 95 %
4. High fever (41 - 42°C)
5. Nasal discharge and excessive salivation
6. Punched out erosions in the mouth (Fig. 47)
7. Loss of appetite and depression
8. Abdominal pain (grunting, arched back)
9. Constipation followed by bloody diarrhoea and straining
10. Dehydration and rough hair coat
11. Marked debility
12. Abortion
13. The classical "milk fever position" in cattle

Postmortem findings :

1. Punched out erosions in the oesophagus
2. Edema or emphysema of the lungs
3. Haemorrhage in the spleen, gallbladder and urinary bladder
4. Haemorrhagic or ulcerative lesions in the omasum
5. Congested abomasum filled with bloody fluid. Ulcers may also be observed.
6. Severe congestion and haemorrhage in the intestine and enlarged and necrotic Peyer's patches (Fig. 48)
7. Last portion of the large intestine and rectum are haemorrhagic showing "tiger stripping" of longitudinal folds
8. Enlarged and edematous lymph nodes
9. Emaciated carcass

Judgement : The carcass derived from a feverish and debilitated animal showing the sign of acute disease on antemortem examination should be *condemned.* In the areas free of RP and in zones where final stages of eradication exist, the animals are also condemned. In endemic zones, if acute symptoms of the disease are not present during clinical examination, the carcass *may have limited distribution.* In areas affected with outbreak which are protected by vaccination, *heat treatment of meat is suggested* if economically worthwhile. The affected organs are *condemned.*

Remarks : Rinderpest virus is sensitive to environmental changes and is destroyed by heat, drying and great number of disinfectants.

Differential diagnosis : Bovine viral diarrhoea, malignant catarrhal fever, infectious bovine rhinotracheitis, bluetongue, coccidiosis, foot and mouth disease, vesicular and necrotic stomatitis and bovine papular stomatitis. Vesicular diseases do not have accompanying haemorrhage and blisters should be differentiated from erosions (ulcers) seen at RP.

Fig. 47: Rinderpest. Erosions on the dental pad and the hard palate which resemble FMD.

Fig. 48: The mucosal surface of Peyer's Patches showing necrosis and congestion.

Vesicular stomatitis (VS)

This is a viral disease of *ruminants, horses* and *swine* characterized by vesicular lesions of the mouth, feet and teats. VS virus has two immunologically distinct serotypes, Indiana and New Jersey.

Transmission : In susceptible animals, contamination of pre-existing abrasions with saliva or lesion material, by ingestion of contaminated pasture or during milking within dairy herds. Mechanical transmission by biting arthropods is also a possibility.The virus is isolated from mites, tropical sand flies and mosquitos.

Antemortem findings :

1. Fever
2. Mouth lesions in cattle and horses
3. Vesicles tend to disappear quickly and only papules may be

seen in cattle outbreaks.
4. Marked weight loss and cessation of lactation in dairy cows.
5. Chewing movements and profuse salivation
6. Refuse food but eagerly accept water
7. Horses rub lips on edges of mangers
8. Foot lesions occur in about 50 % cases in cattle.
9. Lameness
10. Teat lesion may occur in all species.

Postmortem findings :

1. The skin and mucous membrane lesions resemble the lesions of other vesicular diseases.
2. Secondary bacterial or fungal infections
3. Mastitis

Judgement : The carcass of an animal affected with vesicular stomatitis is *approved* if the disease is not in the acute stage and secondary changes are not present. Parts of the affected carcass and organs are *condemned*. A carcass showing acute changes and systemic lesions is *condemned*. If VS is not confirmed by laboratory examination, the judgement will be the same as for the FMD.

Differential diagnosis : Foot and mouth disease, swine vesicular exanthema, vesicular disease, bovine papular stomatitis

The mouth and muzzle lesions: Bovine viral diarrhoea, rinderpest, mycotic stomatitis, photosensitization and Potomac valley fever in horses

Teat lesions: Cowpox, pseudo-cowpox, pseudo-lumpy skin disease and bovine herpes mammillitis

Fig. 49: Vesicular stomatitis. Tongue lesions.

Malignant catarrhal fever (MCF)

An acute viral disease of *cattle, deer, bison* and *buffalo* characterized by inflammation of mucous membranes of the nose, eyes, corneal opacity, profuse nasal discharge and enlargement of lymph nodes. MCF is arbitrarily divided into peracute, intestinal, head-eye and mild forms according to antemortem findings. It is not communicable to man.

Transmission : Close contact between cattle and wildebeest (gnu, antelope), by common use of drinking troughs or by direct contact between cattle and newborn wildebeest and placenta of parturient dams. In American or European MCF, cattle are infected from sheep.

Antemortem findings :

1. Incubation: 9 - 44 days
2. Morbidity is low and mortality is high

3. Increased temperature
4. Bilateral ocular and nasal discharges
5. Dyspnea and cyanosis
6. Loss of appetite
8. Encrustation of muzzle and eczema of the perineum, scrotum and udder
9. Erosions on the lips, tongue, gums, soft and hard palate
10. Swollen reddened eyelids, corneal opacity and conjunctivitis (Fig. 50)
11. Photophobia associated with corneal opacity and blindness
12. Reluctance to swallow because of oesophageal erosions and drooling
13. Enlarged body lymph nodes
14. Rarely, uncoordinated movements and shivering

Postmortem findings :

1. Lesions are not present in acute cases
2. Crater like erosions of the nose, mouth, conjunctiva, oesophagus and gastrointestinal tract
3. Lungs may be congested, swollen or emphysematous
4. White areas in the kidneys
5. Swollen and reddened abomasal folds
6. Intestinal edema and petechial haemorrhage
7. "Tiger striping" in the distal colon (Fig. 51)
8. Enlarged and reddened lymph nodes
9. Dehydrated and emaciated carcass

Judgement : In the early stages of the disease, when fever, emaciation and systemic signs are lacking, the carcass of the affected animal may be *approved as inferior* meat. Otherwise, when fever, emaciation and systemic signs are present, the entire carcass and viscera are *condemned*. The condemned material may be used for rendering.

Differential diagnosis : Bluetongue, rinderpest, bovine viral diarrhoea/mucosal disease, foot and mouth disease, vesicular stomatitis

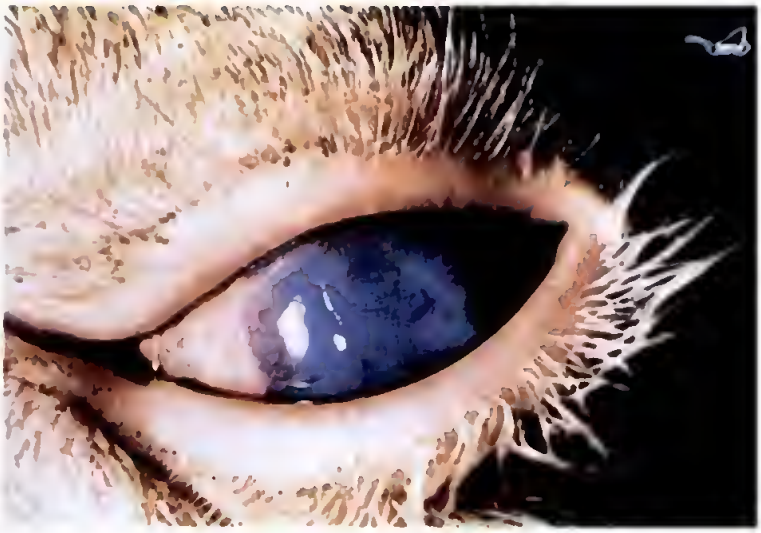


Fig. 50: Malignant catarrhal fever. Early stages of corneal opacity, conjunctivitis and the reddening of the eye lids.

Fig. 51: Malignant catarrhal fever. "Tiger striping" in the distal colon.

Rift valley fever (RVF) (see Chapter 5)

Rabies

This is an acute infectious viral disease of the central nervous system in mammals.

Transmission : It is usually transmitted through the saliva by a bite from a rabid animal, commonly the dog or jackal. Man is infected the same way.

Antemortem findings :

Furious form

1. Incubation from 2 weeks to 6 months or longer
2. Restlessness
3. Aggressive, may attack other animals
4. Sexual excitement
5. Bellowing
6. Paralysis and death

Paralytic form

7. Sagging and swaying of the hind quarters
8. Drooling and salivation
9. The tail is held to one side
10. Tenesmus or paralysis of the anus
11. Paralysis
12. The animal falls to the ground
13. Death after 48 hours of decubitus

Postmortem findings : Possible inflammation of gastrointestinal mucosa

Judgement : In endemic areas carcasses *may be approved* if the animal was bitten eight days before slaughter and within 48 hours of slaughter. The bite area and surrounding tissue must be *condemned*, and prevention taken to prevent occupational hazards.

Differential diagnosis : Indigestion, milk fever or acetonemia when first seen, foreign body in the mouth, early infectious disease, poisoning

Discussion : In a diseased animal, the virus is found in saliva, salivary gland and nervous tissue. Extreme caution should be instituted in abattoirs in order to prevent occupational hazards. Abattoir personnel can contract the disease through surface contact with infected tissue. Infection does not occur by consumption of meat from a rabid animal.

Slaughter may be prohibited during a quarantine period of 8 months following exposure to the disease. An animal suspected of having rabies should be placed under a "Held tag". The warning sign should read "The animal is not to be handled". Any person who was in touch with the animal should thoroughly wash his/her hands with strong soap and/or disinfectant. If possible, the wound should be opened to encourage bleeding in order to flush out the virus and expose the deeper area of the wound. Tincture of iodine (up to 0.001 % aqueous solution of iodine or ethanol 43.70%) should be further applied.

Lumpy skin disease

Acute pox viral disease of *cattle* manifested with sudden appearance of nodules on the skin.

Transmission : Insect vectors by direct and indirect transmission. Seasonal and geographic distribution.

Antemortem findings :

1. Incubation: 4 - 14 days

2. Fluctuating fever
3. Diarrhoea
4. Nasal discharge and salivation
5. The first lesion appear in the perineum
6. Various sized cutaneous nodules (Fig. 52) may occur throughout the body
7. Skin lesions may show scab formation
8. Swelling of superficial lymph nodes and limbs, and lameness
9. Infertility and abortion
10. Secondary infection may lead to joint and tendon inflammation

Postmortem findings :

1. Ulcerative lesions in the mucosa of the respiratory and digestive tract
2. Reddish, haemorrhagic to whitish lesions in the lungs
3. Edema (interlobular) and nodules in the lungs (Fig. 53)
4. Heart lesion (endocardium)
5. Thrombosis of skin vessels followed by cutaneous infarction and sloughing.

Judgement : Carcass of an animal showing mild cutaneous lesions and no fever associated with general signs of infection is *conditionally approved* pending heat treatment. The affected parts of the carcass and organs are *condemned*. Carcass of an animal showing, on antemortem examination, generalized acute infection accompanied with fever, is *condemned*.

Differential diagnosis : Allergies, screw-worm myiasis, urticaria, dermatophilosis (streptothricosis), bovine herpes dermophatic infection, cattle grubs, vesicular disease, bovine ephemeral fever, photosensitization, besnoitiosis (elephant skin disease), sweating weakness of calves, bovine farcy and skin form of sporadic bovine lymphomatosis

Fig. 52: Lumpy skin disease. Various sized cutaneous nodules in a severe case of lumpy skin disease.

Fig. 53: Cut surface of the nodules in the parenchyma of the lung and interlobular edema.

Bovine herpes dermophatic disease (BHD)

A herpes virus infection of *cattle* and sometimes *sheep* and *goats* manifested by cutaneous lesions and fever.

Transmission : Biting insects, mechanical milking

Antemortem and postmortem findings :

1. Incubation: 3 - 7 days
2. Morbidity: High in primary infections
3. Fever
4. Cutaneous nodules. At first these are round, then later become flattened and covered with dry scabs (Fig. 54).
5. Hairless skin is normal after the scab falls off.
6. Ulcerative lesions of the teats and udder (Fig. 55)
7. Erosions between the digits

Microscopy reveals intranuclear inclusions and giant cells in the skin.

Judgement : Carcass of an animal affected with BHD is disposed *similar to* an animal affected with *lumpy skin disease*

Differential diagnosis : Dermatophilis infection, cowpox and pseudocowpox, vesicular stomatitis and lumpy skin disease. The latter is differentiated from BHD by enlarged lymph nodes.

Fig. 54: Bovine herpes dermophatic disease. Dried scabs on the skin of the neck.

Fig. 55: BHD. Ulcerative lesions of the teats and udder.

Infectious bovine rhinotracheitis (IBR)

IBR is a highly infectious viral respiratory disease of *cattle, goats* and *pigs* manifested by inflammation of respiratory passages and pustular lesions on the male and female genital organs. Generally four forms of the disease are recognized; the respiratory form, the genital form, the enteric form and the encephalitic form.

Transmission : Respiratory droplet and nasal exudate in the respiratory form of IBR. Obstetrical operations, coitus and licking of genitalia of affected animals in the genital form of disease.

Antemortem findings :

Respiratory form

1. Incubation: 5 - 14 days
2. Fever
3. Nasal and ocular discharge and red, swollen conjunctiva
4. Drop in milk yield
5. Breathing through the mouth and salivation (Fig. 56)
6. Hyperaemia of the nasal mucosa and necrotic areas on the nasal septum
7. Secondary bronchopneumonia
8. Abortion

Genital form

9. Frequent urination and tail elevation
10. Edematous swelling of the vulva and pustule formation on reddened vaginal mucosa
11. Mucoid or mucopurulent exudate in the vagina

Enteric form

12. Severe oral and stomach necrosis in new born animals
13. High mortality

The encephalitic form in calves

14. Depression
15. Excitement
16. High mortality

Postmortem findings :

1. Acute inflammation of the larynx, trachea (Fig. 57) and bronchi
2. Profuse fibrino-purulent exudate in the upper respiratory tract in severe cases
3. Chronic ulcerative gastro-enteritis in feedlot cattle
4. Lung emphysema
5. Secondary bronchopneumonia

Judgement : Carcass of an animal affected with IBR is *approved* if signs of acute infection are not present and the animal is in good body condition.

Differential diagnosis : Pneumonic pasteurellosis, bovine viral diarrhoea, malignant catarrhal fever and calf diphtheria

Fig. 56: Breathing through the mouth and salivation in a bovine affected with IBR.

Fig. 57: IBR. Acute inflammation of the larynx and trachea

Bovine viral diarrhoea (BVD)

This is an infectious viral disease of *cattle* manifested by an active erosive stomatitis, gastroenteritis and diarrhoea.

Transmission : Direct contact with clinically sick or carrier animals, indirect contact with feedstuffs or fomites contaminated with urine, nasal and oral secretions or faeces and contact with aborted fetuses. Transmission through aerosol droplet dispersion or by insect vector may also be a possibility. Virus may persist in recovered and chronically ill cattle which are considered a potential source of infection.

Antemortem findings :

1. Incubation: 1 - 3 days
2. Fever
3. Congestion and erosions in the mucous membranes of the oral cavity
4. Depression and anorexia
5. Cough, polypnea and salivation
6. Dehydration and debilitation
7. Foul-smelling diarrhoea
8. Cessation of rumination
9. Reduced milk supply
10. Abortion in pregnant cows
11. Laminitis
12. Congenital anomalies of the brain (cerebellar ataxia) and arthritis in young calves

Postmortem findings :

1. Shallow erosions present on the entrance of the nostrils, mouth, pharynx, larynx, oesophagus, rumen (Fig. 58), omasum, abomasum (Fig. 59), caecum and less frequently

in Peyer's patches in the small intestine.

2. Erythema of the mucosa with submucosal haemorrhage in the abomasum, small intestine, caecum and colon. Stripped appearance on the caecal and colon mucosa is similar to that seen in rinderpest.

3. Cerebral hypoplasia and cataracts in calves.

Judgement : Carcass and viscera of an animal, which on antemortem examination showed generalized signs of acute infection accompanied with fever and/or emaciation, are *condemned*. Chronic cases of BVD with no systemic involvement have a *favourable judgement* of carcass, viscera and organs.

Differential diagnosis : Malignant catarrhal fever, rinderpest, blue tongue and vesicular diseases. The latter produce vesicles which are not present in BVD. Diseases with no oral lesion nor diarrhoea include salmonellosis, Johne's disease and parasitism.

Fig. 58: BVD. Congestion and erosions in the ruminal mucosa.

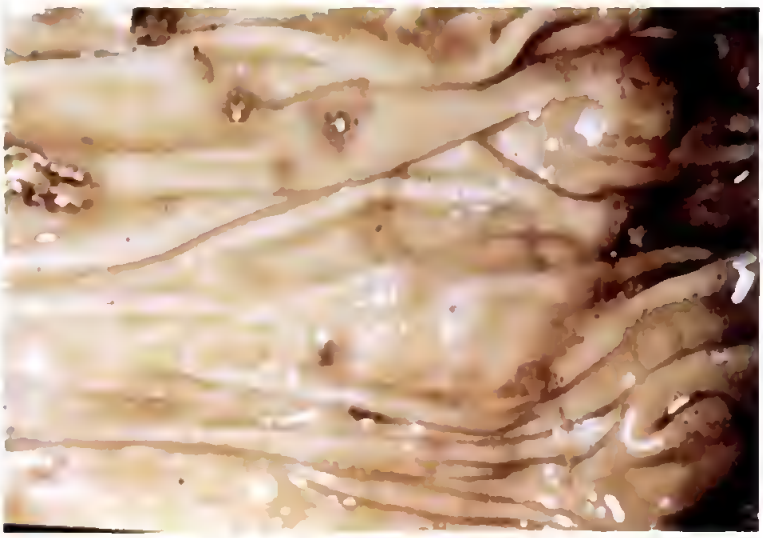


Fig. 59: BVD. Inflammation of the abomasum (abomasitis, gastritis).

Bovine leukosis

Bovine leukosis is a persistent and malignant viral disease of the lymphoreticular system. It occurs in all breeds and in both sexes.

Bovine leukosis is observed in two forms : a) the *sporadic* and b) the *enzootic* form. The sporadic form is rare and occurs in cattle under three years of age. The enzootic form is most commonly found in adult cattle, particularly in cull cows.

Transmission : By small amounts of infected blood (e.g. infected needles, dehorning), vertical transmission from the dam to the calf (3 - 20 % of calves may become infected) and by colostrum or milk (less than 2 %). Insect transmission is also a possibility; higher rates of infection were reported in the summer.

Antemortem findings :

1. Laboured breathing due to heart involvement
2. Persistent diarrhoea following infiltration of the abomasum

wall by neoplastic cells
3. Marked enlargement of several superficial lymph nodes
4. Edema of the brisket and the intermandibular region
5. Paralysis of the hind legs due to tumour compression of the spinal cord
6. Protrusion of the eye as a result of tumour invasion of the orbital cavity
7. Debilitation or emaciation
8. Pale mucosal surface
9. Bloated animal
10. Swelling of the neck when thymus is involved
11. Cutaneous nodules in the terminal stage

Postmortem findings :

1. Lymph node enlargement (clay-like consistency)
2. Enlargement of spleen (splenomegaly)
3. Thin watery blood
4. Neoplastic lesions in the heart (Fig. 60), intestines (Fig. 61) (Virtually all of the organs may be involved.)
5. Ventral edema
6. Enlarged haemolymph nodes

Judgement : Carcass of an animal affected with leukosis (lymphosarcoma) is *condemned*. When a diagnosis cannot be made by postmortem findings, a laboratory diagnosis should be performed. If lymph node hyperplasia is the histological diagnosis, the carcass is *approved* for human consumption. Depending on disease prevalence, leukosis reactors may be totally *approved* or *conditionally approved* pending heat treatment.

Differential diagnosis : Lymphadenitis, lymphoid hyperplasia, hyperplastic haemolymph nodes, pericarditis, enlarged spleen in septicemic conditions, other neoplasms and parasitism.

Fig. 60: Leukosis. Neoplastic mass infiltrating the heart muscle.

Fig. 61: Leukosis. Neoplastic growths in the intestine. Both lesions were histologically confirmed as lymphosarcoma.

Bovine spongiform encephalopathy (BSE, "Mad cow disease")

BSE is a progressive and fatal disease of adult cattle characterized by a progressive degeneration of the central nervous system causing neurological signs in animals. Some scientists

suspect that an unusual and atypical virus-like transmissible agent called a prion is associated with the etiology of BSE. Prion is the term currently used in literature.

Transmission : The ingestion of protein feed supplements prepared from sheep meat or sheep by products contaminated with scrapie virus.

Antemortem findings :

1. Incubation period 2 - 8 years
2. Reduction in milk production
3. Weight loss, while maintaining good appetite
4. Behaviour changes (nervousness and aggressiveness), kicking in the milking parlour

The progressive degeneration of the central nervous system causes neurologic signs:

5. Apprehension, teeth grinding
6. Tremors and abnormal ear position
7. Abnormal posture and disorientation
8. Incoordination and stiff gait
9. Paresis
10. Recumbency and death

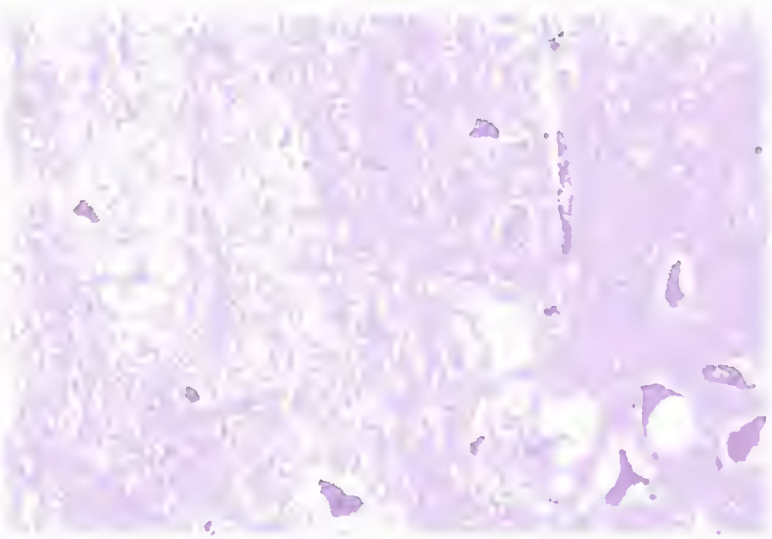


Fig. 62: BSE. Degenerative lesion in the cerebral cortex.

Diagnosis can be confirmed only on the postmortem histological examination of brain tissue. Microscopic lesions include degenerative lesions of the cerebral cortex (Fig. 62), medulla and central grey matter of the midbrain.

Judgement : Carcass is *condemned*.

Differential diagnosis : Rabies, listeriosis, bovine pseudorabies (mad itch), other brain infections in cattle, the nervous type of acetonemia, hypocalcemia, hypophosphatemia and hypomagnesemic tetany

Discussion : The first reported cases of this disease were in dairy cows in 1987 from different locations in the United Kingdom. The disease is now also recognized in some other countries in and outside Europe. BSE belongs to a group of human and animal diseases classified as transmissible spongiform encephalopathies. Significant human diseases of this group are Kuru and Creutzfeldt-Jacob's disease. Scrapie, which affects sheep and goats also belongs in this group.

Researchers are trying establish if BSE and scrapie have the same causative agent, and if the modified form of the scrapie agent is also a possible causative agent of BSE. Prions are also the causative agents of transmissible mink encephalopathy (TME) and of chronic wasting disease (CWD) of mule deer and elk.

BSE affects only adult animals and the incidence within-herd is low. The breed, gender or year and seasons are not associated with the development of this disease, nor is contact with sheep. In order to control this disease, in the U.K. the following actions were taken:

1. Ruminant derived protein is prohibited in all ruminant rations.

2. The consumption of milk from affected animals by humans or animals is also prohibited.
3. Bovine brain cannot be used for human consumption.
4. The mandatory slaughter of all animals manifesting signs of BSE and compensation awarded to the owner.

Diseases caused by Rickettsia and Mycoplasma spp.

Heartwater (Hydropericardium)
"Black dung" when affecting African cattle and buffalo
"Sheep fever" when seen in sheep

Heartwater is an acute, non contagious disease of *cattle, sheep, goats, antelopes* and *wild ruminants*. It is caused by the rickettsial organism *Cowdria (Rickettsia) ruminantium*.

Transmission : Heartwater is transmitted by various species of Amblyomma ticks. Transstadial transmission of the organism occur in vector ticks.

Antemortem findings :

Peracute form

1. Incubation 14 - 28 days
2. Fever
3. Diarrhoea
4. Convulsions and death

Acute form

5. Fever up to 41.7°C
6. Rapid breathing
7. Lack of appetite, depression and listlessness

Nervous signs include

8. Twitching of the eyelids
9. Protrusion of the tongue
10. Champing of the jaw
11. Walking in circles
12. Paddling with legs in recumbent animals
13. Opisthotonos and convulsions

Postmortem findings :

1. Hydropericardium
2. Hydrothorax
3. Pulmonary edema and ascites
4. Haemorrhagic gastroenteritis
5. Enlarged liver, spleen and lymph nodes
6. Haemorrhage in the abomasum and intestine
7. Edema and haemorrhage of the brain

Judgement : Carcass of an animal affected with heartwater is *condemned* in the acute stage of the disease. In a chronic case, the carcass *may be approved* if adequately bled and muscles are wholesome in colour and texture. The affected organs are *condemned.*

Differential diagnosis : Peracute form of heartwater should be differentiated from anthrax. The acute nervous form of the disease is differentiated from tetanus, rabies, cerebral trypanosomiasis, strychnine poisoning, piroplasmosis, theileriosis, lead and organophosphate poisoning, parasitism, arsenical poisoning and poisoning with certain plants.

Fig. 63: Heartwater. Cowdria ruminantium in bovine brain smear (arrow).

Q fever (Queensland fever, Nine mile fever, American Q fever, Australian Q fever)

Q fever is a disease of cattle, sheep, goats, donkeys, camels, fowl, dogs, cats, pigeons and humans. It is caused by *Coxiella burnetii*. Q fever is an occupational disease of livestock personnel, farmers and laboratory personnel.

Transmission : Ticks spread infection to cattle which develop mild disease. The faeces deposited on animal hide by ticks may be the source of infection for humans. Q fever is also transmitted by inhalation or dust contaminated with infected animal secreta or excreta. Healthy animals may serve as a carrier and shed the organism in milk, urine, faeces, placenta and fetal fluids. They harbour the infection and no clinical signs are observed. Contaminated meat and water are further means of infection spread.

In field cases there are no clinical signs of this disease. In the

disease produced by the inoculation of cows via the udder the clinical signs may include:

1. Acute mastitis
2. Loss of appetite and depression
3. Serous nasal and lacrimal discharge
4. Difficult breathing
5. Atony of the rumen
6. Abortion in pregnant cows

No gross lesions are reported in cattle.

Discussions : *Coxiella burnetii* is highly resistant and was isolated from farm soil 6 months after the removal of animals. It may persist in the udder up to 3 years. The temperatures of milk pasteurisation (in bulk at 63°C for 30 minutes or the common method at 72°C for 15 seconds) kill this agent in milk. Vaccination will reduce shedding of organisms in milk.

This disease in *humans* has a sudden onset and is characterized by loss of appetite, weakness and generalized malaise lasting from 1 - 2 weeks. Pneumonia may also be present. Death may be caused by endocarditis in older people. More severe symptoms of Q fever are noticed.

Contagious bovine pleuropneumonia

This is an acute, subacute or chronic highly infectious disease of cattle caused by *Mycoplasma mycoides var. mycoides.*

Transmission : Aerosol and droplet infection from the infected animals. The recovered animal called "lungers" act as carriers and shedders, especially under stress.

Antemortem findings :

1. Incubation: acute 10 - 14 days, chronic 3 - 6 months
2. Morbidity: 90 % in susceptible cattle
3. Mortality: 10 - 50 %
4. Fever
5. Depression
6. Lack of appetite and loss of weight
7. Coughing on exercise
8. Shallow rapid respiration, grunting and gurgling
9. Extended neck, lowered head and open mouth
10. Arched back and outward rotated elbow
11. Arthritis in young animals

Postmortem findings :

1. Fibrinous inflammation of the pleura (pleuritis)
2. Straw coloured fluid in the thorax (Fig. 64)
3. Lobar pneumonia with red hepatization, marbled appearance of lung lobules (Fig. 65) due to thickening of interlobular septae and interlobular pulmonary edema
4. Enlarged mediastinal lymph nodes
5. Walled-off sequestra formation in chronic cases
6. Haemorrhage in the heart
7. Arthritis and tenosynovitis

Judgement : Carcass of an animal affected with contagious bovine pleuropneumonia is *condemned* if the disease is associated with fever, inadequate bleeding of carcass, serous infiltration of the brisket and emaciation. Recovered animals showing no generalized signs of the disease are *approved* and the affected organs are *condemned.*

Differential diagnosis : Shipping fever (Pasteurellosis), East coast fever, foreign body pneumonia, IBR, tuberculosis, chlamidial

infections and lungworms

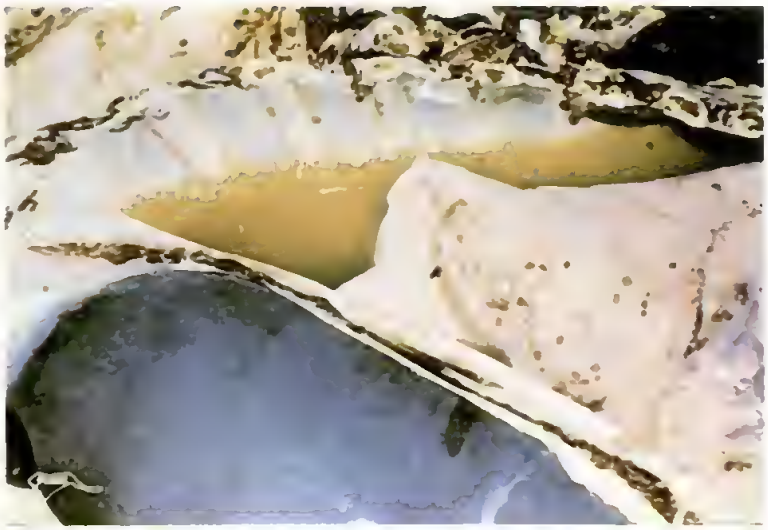


Fig. 64: Contagious bovine pleuropneumonia. Straw coloured fluid in the thorax and partial lung hepatization.

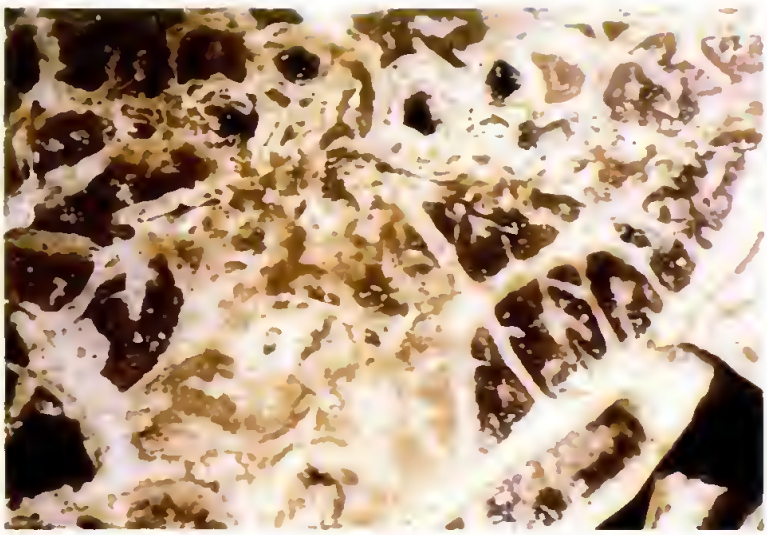


Fig. 65: Contagious bovine pleuropneumonia. Lobar pneumonia with red hepatization and marbled appearance of lung lobules.

Diseases caused by bacteria

Black quarter (Black leg)

Black quarter is an acute infectious disease of cattle and sheep manifested by severe inflammation of the muscle with high mortality. It is caused by *Clostridium chauvoei.*

Transmission : The organisms of blackleg are found in the soil. During grazing, organisms may enter the digestive tract of a susceptible animal. *Clostridium chauvoei* is also found in the digestive tract of healthy animals. In sheep the agent is transmitted through wounds at shearing, docking and castration and during lambing in ewes.

Antemortem findings :

1. High fever (41°C)
2. Lameness
3. Loss of appetite
4. Discoloured, dry or cracked skin
5. Stiff gait and reluctance to move
6. Crepitating swellings often on the hips and shoulder
7. In sheep gaseous crepitation cannot be felt before death

Postmortem findings :

1. Laying on one side with affected hind leg stuck out. Commonly seen in cattle
2. Bloating of carcass and blood stained frothy exudates from the nostrils and anus
3. Dark red to black muscle of the loin, back or leg (Fig. 66)
4. Spongelike bubbly appearance of the muscles with a peculiar rancid odour
5. Yellowish, gelatinous subcutaneous tissue and associated gas

bubbles
6. Blood stained fluid in body cavities

Fig. 66: Black leg. Dark-red skeletal muscle of a heifer showing haemorrhage, necrosis, edema and emphysema.

Judgement : Carcasses of animals affected with black leg should be *condemned*. It is prohibited to slaughter and dress an animal diagnosed with this disease at antemortem examination.

Differential diagnosis : Other acute Clostridial infections, lightning strike, anthrax, bacillary haemoglobinuria, lactation tetany, extensive haemorrhage and acute lead poisoning.

Discussion : Black leg is worldwide in distribution. Well nourished animals are more frequently affected. It is also more commonly seen in grass fed animals than in stall fed animals. Clostridia are soil-borne organisms which cause disease by releasing toxins. Specific antitoxin and antibiotics are rarely effective in the treatment of this disease. An adequate preventive vaccination program may be the most effective method in protecting the animals from black leg.

Botulism

Botulism is a disease manifested by progressive muscular paralysis. It is seen in humans, animals, birds and fish and is caused by various strains of *Clostridium botulinum*.

Transmission : Decomposed flesh and bones are the source of infection for animals. Incubation period 12 - 24 hours. However, 2 hours up to 14 days incubation period has been recorded.

Antemortem findings :

In cattle and horses

1. Restlessness
2. Knuckling and incoordination
3. Paralysed tongue and drooling of saliva
4. Sternal recumbency
5. Progressive muscular paralysis from hindquarters to frontquarters, head and neck (Fig. 67)

In sheep

6. Serous nasal discharge and salivation
7. Abdominal respiration
8. Stiffness upon walking and incoordination
9. Switching of the tail on the side
10. Limb paralysis and death

In pigs

11. Lack of appetite, refusal to drink and vomiting
12. Pupillary dilatation
13. Muscular paralysis

Postmortem findings : Foreign material in fore-stomachs or stomachs may be suggestive of botulism.

Judgement : Total condemnation of carcass because of human hazards.

Differential diagnosis : Parturient paresis, paralytic rabies, equine encephalomyelitis, ragwort poisoning in horses, miscellaneous plant poisoning.

In sheep - louping ill, hypocalcemia and some cases of scrapie.

Discussion : Cl. botulinum is found in the digestive tract of herbivores. Soil and water contamination occurs from faeces and decomposing carcasses. The proliferation of Cl. botulinum organisms may also occur in decaying vegetable material. Sporadic outbreaks of botulism are reported in most countries. Outbreaks of botulism in cattle and sheep in Australia, Southern Africa and the Gulf coast area of the United States are associated with phosphorus deficient diets and ingestion of carrion. Cattle, sheep and rarely swine are susceptible to this disease. Dogs and cats are resistant.

Cl. botulinum produces neurotoxin which causes functional paralysis. Seven strains of this organism (" A through G ") are distinguished according to immunological differences. The diseases caused by various strains of this agent are frequently regarded as a separate entity owing to some of their prominent signs. Names such as "*Bulbar paralysis in cattle*", "*Lamsiekte in sheep*" in South Africa (meaning lame sickness), and "*Limberneck in poultry*" are often used. Cl. botulinum is often found in anaerobic conditions of deep wounds. It produces neuroparalytic exotoxins which cause symptoms of the disease. This organism will grow and produce toxins if the temperature is between 10 - 50°C, pH above 4.6, water activity (AW) above 0.93 and anaerobic conditions exist. Fresh meats are implicated with less than 10 % of botulism outbreaks. The major sources of this organism are fish, home cured meats, home canned vegetables and fruit. Eggs, milk and their products are rarely the cause of an outbreak. Most

frequently, raw, insufficiently cooked foods or food not fully salted, cured, dried or smoked are implicated. Botulism toxins are heat labile and food suspected of having the organism should be boiled before serving.

In *man* the signs of the disease are weakness, dizziness, blurred or double vision, dilatation of pupils, dry mouth, difficulties in breathing and speech, progressive muscular weakness, respiratory failure and death. Pneumonia may be a complication associated with botulism in man.

Fig. 67: Botulism. Sternal recumbency. Muscular paralysis of hind and front quarters.

Malignant edema

Malignant edema is a bacterial disease of cattle, sheep, goats, swine, horses and poultry. It is caused by *Clostridium septicum* and is manifested by wound infection. The infection is commonly soil-borne. Deep wounds associated with trauma provide ideal condition for the growth of this agent.

Antemortem findings :

1. Fever 41 - 42°C
2. Depression and weakness
3. Muscle tremor and lameness
4. Soft doughy swelling and erythema around the infection site

Postmortem findings :

1. Gangrene of the skin in area of infection site
2. Foul putrid odour is frequently present
3. Gelatinous exudate in the subcutaneous and intramuscular connective tissue
4. Subserosal haemorrhage
5. Accumulation of sero-sanguineous fluid in body cavities
6. Muscle tissue is dark-red but has little or no gas

Judgement : Carcasses of animals affected with malignant edema are *condemned.*

Differential diagnosis : Blackleg. In malignant edema the muscle is not involved and the wound site is noted. Anthrax in pigs. Subcutaneous edema in the throat region is present.

Tuberculosis

Tuberculosis is a chronic disease of many animal species and poultry caused by bacteria of the genus *Mycobacterium.* It is characterized by development of tubercles in the organs of most species. Bovine tuberculosis is caused by *Mycobacterium bovis.* It is a significant zoonotic disease.

Transmission : An infected animal is the main source of

transmission. The organisms are excreted in the exhaled air and in all secretions and excretions. Inhalation is the chief mode of entry and for calves infected milk is an important source of infection. When infection has occurred tuberculosis may spread: a) by primary complex (lesion at point of entry and the local lymph node) and b) by dissemination from primary complex.

Antemortem findings :

1. Low grade fever
2. Chronic intermittent hacking cough and associated pneumonia
3. Difficult breathing
4. Weakness and loss of appetite
5. Emaciation
6. Swelling superficial body lymph nodes

Postmortem findings :

1. Tuberculous granuloma in the lymph nodes of the head, lungs (Fig. 68), intestine and carcass. These have usually a well defined capsule enclosing a caseous mass with a calcified centre. They are usually yellow in colour in cattle, white in buffaloes and greyish white in other animals.
2. Active lesions may have a reddened periphery and caseous mass in the centre of a lymph node.
3. Inactive lesions may be calcified and encapsulated
4. Nodules on the pleura and peritoneum
5. Lesions in the lungs (Fig. 69), liver, spleen, kidney
6. Bronchopneumonia
7. Firmer and enlarged udder, particularly rear quarters
8. Lesions in the meninges, bone marrow and joints

The diagnosis may be confirmed by making a smear of the lesion and staining with Ziehl-Neelsen. The TB bacterium is a very small

red staining bacillus.

Fig. 68: Tuberculous granuloma in the mediastinal lymph nodes. M. bovis was isolated.

Fig. 69: Lesion of tuberculosis in the lungs.

Discussion : Mycobacteria invade cattle by respiratory (90 - 95 %) and oral routes (5-10 %). Congenital infection in the bovine fetus occurs from an infected dam. Tuberculosis lesions can be

classified as *acute miliary*, *nodular lesions* and *chronic organ* tuberculosis. Young calves are infected by ingestion of contaminated milk. The incidence of human tuberculosis caused by Mycobacterium bovis has markedly dropped with the pasteurization of milk. It also has dropped in areas where programs of tuberculosis eradication are in place. Man is susceptible to the *bovine type*. In cattle, lesions of tuberculosis caused by the *avian type* are commonly found in the mesenteric lymph nodes. Tuberculosis in small ruminants is rare. In pigs the disease may be caused by the *bovine and avian types*. Superinfection is specific in cattle.

Judgement : Carcass of an animal affected with tuberculosis requires additional postmortem examination of the lymph nodes, joints, bones and meninges. It is suggested that the Codex Alimentarius judgement recommendations for cattle and buffalo carcasses be followed.

Carcasses are *condemned*

i) where an eradication scheme has terminated or in cases of residual infection or re-infection
ii) in final stages of eradication - natural prevalence low
iii) during early stages in high prevalence areas

Carcass of a reactor animal without lesions *may be approved* for limited distribution. If the economic situation permits, this carcass should be *condemned*. *Heat treatment* of meat is suggested during early and final stages of an eradication programme: in low and high prevalence areas where one or more organs are affected, and where miliary lesions, signs of generalization or recent haematogenous spread are not observed. If the economical situation permits, then the carcass is *condemned*.

In some countries, the carcass is *approved* if inactive lesions (calcified and/or encapsulated) are observed in organs and without

generalization in lymph nodes of carcass.

Differential diagnosis : Lung and lymph node abscess, pleurisy, pericarditis, chronic contagious pleuropneumonia, actinobacillosis, mycotic and parasitic lesions, tumours, caseous lymphadenitis Johne's disease, adrenal gland tumour and lymphomatosis

Johne's disease (Bovine paratuberculosis)

Johne's disease is a chronic, infectious bacterial disease of adult wild and domestic ruminants such as cattle, sheep, and goats. It is characterized by the thickening and corrugation of the wall of the intestine, gradual weight loss and chronic diarrhoea and is caused by *Mycobacterium paratuberculosis*.

Transmission : Ingestion of faeces harbouring Mycobacterium paratuberculosis

1. The agent is persistent in soil, pasture, manure and stagnant water for prolonged period.
2. Carrier animals, so called "faecal shedders", are the most important source of infection.
3. Ingestion of organism. Calves may become infected from a nursing infected dam.
4. Transmission with semen and in-utero are minor source of infection

Antemortem findings :

1. Incubation period 2 - 3 years with range from 6 months to 15 years.
2. Indifferent animal which stops eating at the end of the disease
3. Gradual and chronic weight loss and emaciation
4. Rough hair coat and dry skin

5. Non responsive diarrhoea with watery fluid faeces
6. Submandibular edema ("bottle jaw")
7. Reduced milk production
8. Mastitis and infertility
9. Debility and death

Postmortem findings :

1. Thickened and corrugated intestinal mucosa (Fig. 70)
2. Enlarged caecal lymph nodes

Judgement : Carcass of an animal affected with Johne's disease is *approved* when generalized systemic signs of disease are not present. A poor, thin and slightly moist carcass should be held in the chiller and assessed after 24 or 48 hours. If the dryness and setting of the carcass improves during this time it can be *released*. The carcass with associated edema and emaciation is *condemned*.

Differential diagnosis : Other causes of diarrhoea and weight loss, malnutrition, chronic salmonellosis, parasitism (e.g. Ostertagiasis), winter dysentery, Bovine Viral Diarrhoea (BVD), "hardware" disease, coccidiosis, liver abscesses, kidney disease, inflammation of the heart and its sac, toxic inflammation of the intestine caused by arsenic, plants and mycotoxicosis and neoplasm.

Fig. 70: Johne's disease. Thickened and corrugated intestinal mucosa.

Leptospirosis

Leptospirosis is an important and relatively common disease of domestic and wild animals and humans. In cattle, it is manifested by interstitial nephritis, anaemia and mastitis and abortion in most species. *Leptospira spp.* are the causative agents.

Transmission : Animals contract the disease by eating and drinking leptospira-contaminated urine, water, or by direct contact of broken skin or mucous membranes with mud, vegetation or aborted fetuses of infected or carrier animals. Recovered animals and animals with unapparent (subclinical) leptospirosis frequently excrete billions of leptospiras in their urine for several months or years.

Antemortem findings :

Acute and subacute forms

1. Transient fever
2. Loss of appetite
3. Lactating cows may stop milking
4. Mastitis
5. Milk may be yellow, clotted and frequently blood stained

Severely affected animals

6. Jaundice and anaemia
7. Pneumonia
8. Abortion with frequent retention of the placenta (afterbirth)

Severe illness in young calves may be associated with yellowish discoloration of mucous membranes and reddish-brown urine before death. The chronic form has mild clinical signs and only abortion may be observed. If meningitis occurs, the animal may show incoordination, salivation and muscular rigidity.

Postmortem findings :

1. Anaemia and jaundice
2. Subserosal and submucosal haemorrhage
3. Ulcers and haemorrhages in the abomasal mucosa
4. Rarely pulmonary edema or emphysema
5. Interstitial nephritis (Fig. 71)
6. Septicaemia

Fig. 71: Leptospirosis. Interstitial nephritis in a bovine.

Judgement : Carcass of an animal affected with acute leptospirosis is *condemned.* A chronic and localized condition may *warrant an approval* of the carcass.

Differential diagnosis : Acute and subacute forms to be differentiated from babesiosis, anaplasmosis, rape and kale poisoning, bacillary haemoglobinuria, post parturient haemoglobinuria and acute haemolytic anaemia in calves. The presence of blood in the milk is a characteristic clinical sign which will differentiate leptospirosis from other infectious diseases.

Discussion : Leptospirosis is a zoonosis and is also an occupational hazard for farmers, veterinarians and butchers.

Human infection may occur by contamination with infected urine and urine contents. The bacteria may be also found in milk in acute cases, however, it does not survive for long period of time in milk. Pasteurization will also kill leptospiras. They can survive for months in moist and humid environments, particularly in swamps, ponds and streams or poorly drained pastures.

Brucellosis (contagious abortion, Bang's disease)

Brucellosis of cattle is an infectious, contagious disease caused by *Brucella abortus* and is characterized by abortion in late pregnancy and a high rate of infertility. *B. melitensis* affects goats, *B. ovis* sheep and *B. suis* swine. *B. abortus* may occur in horses.

Transmission : An uninfected animal may become infected with Brucella organisms by contaminated feed, pasture, water, milk, by an aborted fetus, fetal membranes and uterine fluid and discharges. The disease may also be spread by dogs, rats, flies, boots, vehicles, the milking machine and other equipment used in the barn. The Brucella organism may be occasionally shed in urine.

Antemortem and postmortem findings :

In cattle

1. Abortion in non vaccinated pregnant cows in the last 3 - 4 months of pregnancy
2. Occasional inflammation of testes and epididymis
3. Swelling of scrotum (one or both sacs)
4. Edematous placenta and fetus
5. Hygromas on the knees (Fig. 72), stifles, hock and angle of the haunch, and between the nuchal ligament and the primary thoracic spines.

In sheep

6. Fever, increased respiration and depression
7. Inferior quality of semen in rams
8. Edema and swelling of scrotum (see Fig. 163A in Chapter 5)
9. In chronic stage enlarged and hard epididymis, thickened scrotal tunics and frequently atrophic testicles
10. Infertility in rams and abortion in ewes

Judgement : Cattle and horse carcasses affected with brucellosis are *approved* (after removal of affected parts), as Brucella bacteria remain viable for only a short period in the muscles after slaughter. In acute abortive form (after the miscarriage), cattle carcasses are *condemned*. Pig, sheep, goat and buffalo carcasses require *total condemnation*. *Heat treatment* may be recommended in some areas for these species due to economic reasons. Affected part of the carcass, udder, genital organs and corresponding lymph nodes must be *condemned*.

Reactor animals should be carefully handled during slaughter and dressing procedures. Gloves and goggles should be worn when known reactors are being slaughtered and hygroma lesions should be sprayed liberally with 1 % lactic acid at meat inspection.

Differential diagnosis : Causes of abortion in cattle, IBR, vibriosis, leptospirosis, trichomoniasis, mycoplasma infections, mycosis, nutritional and physiological causes.

Discussion : Brucella organisms have only a short life in the muscles of slaughtered animals. They are destroyed by lactic acid. While slaughtering and dressing the reactors, a hook should be used in handling the uterus and udder. Employees in close contact with infected animals should wear gloves and avoid accidental cuts.

In *humans*, brucellosis is called "Undulant Fever". The general population is not at risk with this disease if high levels of hygiene and sanitation are practised. Pasteurized milk is brucella-free. Affected humans will suffer from intermittent high fever, headache and generalized malaise.

Brucellosis is an important zoonosis in particular in rural areas in developing countries and is an important occupational hazard for veterinarians, meat inspectors, farmers, animal health inspectors and butchers.

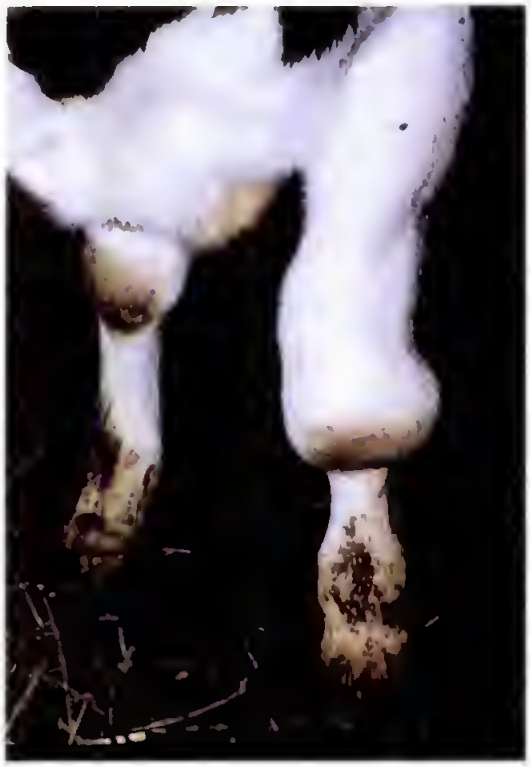


Fig. 72: Brucellosis. Hygromas on the knee joints. This condition may be a sequel to Brucella abortus infection.

Anthrax

Anthrax is a peracute disease of ruminants manifested with septicemia, sudden death and tarry blood from the body openings of the cadaver. It is caused by *Bacillus anthracis*.
Transmission : Man may contract anthrax by inhalation, ingestion and through a wound in the skin. Biting flies have been shown to be transmitters.

Antemortem findings :
The peracute and acute forms in cattle and sheep are without clinical signs. Death may follow in the acute form after 1 - 2 hours of illness. The acute form lasts about 48 hours.

In pigs and horses this disease is usually localized and chronic and is often characterized by swelling around the throat and head.

Antemortem findings in pigs :
1. Incubation 1 - 2 weeks
2. Edematous swelling of the throat and neck
3. Swallowing and breathing difficulties
4. Death due to choking or toxaemia
5. Septicemia is not observed.

Postmortem findings :

1. Dark-tarry blood discharge from body orifices
2. Absence of rigor mortis
3. Haemorrhage of the mucous and serous membranes, lymph nodes and subcutaneous tissue
4. Enlarged spleen
5. Severe haemorrhagic enteritis
6. Degeneration of the liver and kidneys
7. Bloating and rapid decomposition of carcass
8. Localized lesions in the intestine of pigs (dysentery)

Diagnosis of anthrax is carried out by direct microscopic examination of tissues and fluids (Fig. 73).

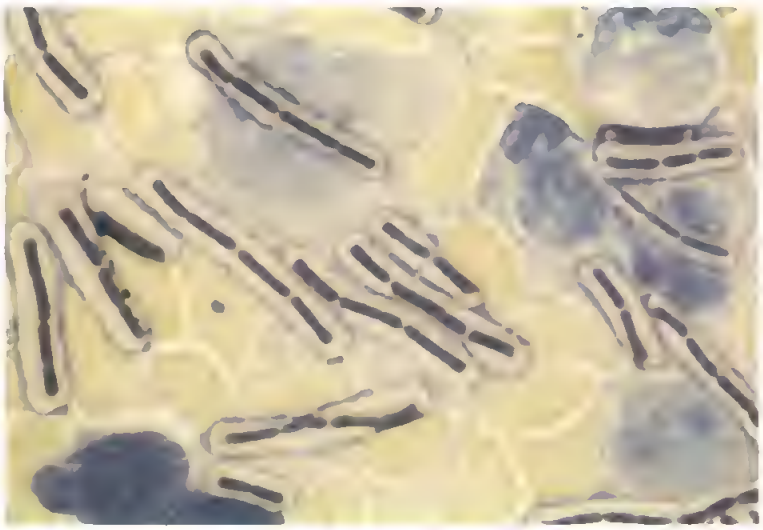


Fig. 73: Anthrax. Toluidine blue stain. Bacillus anthracis in a bovine spleen. Anthrax bacilli in tissue seen in short chains surrounded by a common capsule.

Judgement : *Condemnation* of the carcass and its parts by burning or burial. If disposed by burial, the carcass should be buried at least 6 feet below ground. The site should be surrounded by a foot thick layer of quicklime.

Differential diagnosis : Peracute blackquarter and septicaemic form of other diseases. In splenic enlargement as seen in babesiosis, anaplasmosis and leucosis, spleen consistency is firm. In anthrax, the spleen is soft and upon incision the pulp exudes like thick blackish-red blood.

Discussion : If an animal has died from an unknown cause in an abattoir's pen or in the stockyard, a blood smear from the tip of the ear should be examined to eliminate anthrax as a cause of death. All measures should be taken to prevent further contact with the carcass. The orifices of the nose, vulva and anus should be packed with cotton swabs to eliminate further spillage of discharge. The carcass must not be opened. Due to insufficient

oxygen supply in the closed carcass, spores of B. anthracis will not be formed and the organism will be killed. The spilled discharge is firstly removed by drying with sawdust and sand and is then destroyed together with the carcass. The carcass is wrapped in thick plastic sheets and destruction is performed under the supervision of an appropriate government official.

An open carcass facilitates exposure of B. anthracis to air and consequently, spores are formed within a few hours. Anthrax spores are resistant to heat and disinfectants and may survive in a suitable environment for years.

The abattoir's pen or stockyard area suspected of being in contact with an anthrax animal should be disinfected with *10 % NaOH* or *5 % formaldehyde* and cleaned. This cleaning should also include the cattle trucks or cars used for the transportation of infected animals. All personnel that were in contact with anthrax or that handled contaminated material, are also subjected to decontamination. The arms and hands should be washed with liquid soap and hot water. After they have been rinsed, they should be immersed for about one minute, in an *organic iodine solution* or 1 p.p.m. solution of *mercuric perchloride* or other acceptable agents. This is followed by a potable water rinse. Clothing of the personnel involved should also be cleaned and thoroughly disinfected by boiling.

If the carcass is discovered on the killing floor, all operations must cease. The carcass and its parts including hides, hooves, viscera and blood must be condemned and destroyed. The carcasses which have been dressed by the same abattoir employees prior to or after the affected carcass must also be condemned and destroyed. Those carcasses which had been dressed before the affected carcass may have a second option of being salvaged with sterilization. They must be boiled for a minimum of 3 hours if contamination occurred with blood splashes. If impractical, these carcasses may

be used for "canned meat" for which heat treatment is recommended.

Disinfection of equipment used for the dressing of a diseased carcass as well as the infected abattoir area, should be done with *5 % solution of sodium hydroxide (NaOH)*. This disinfectant is used because of its action on fat and grease removal. *Heat* in the form of a blowtorch can be used for disinfecting buildings.

Salmonellosis in bovine

Salmonellosis is a disease which occurs in all animals and humans. In animals, salmonellosis is characterized clinically by one of three syndromes: a) *peracute septicemic form*, b) *acute enteritis* or c) *chronic enteritis*.

The young, old, debilitated and stressed animals are at greater risk. More then 200 antigenically different serotypes of Salmonella have been identified and all of these possess pathogenic potential. The most frequently identified serotypes of the organisms which cause the disease in cattle are *S. typhimurium, S. dublin, S. muenster* and *S. newport*. Salmonellosis in stressed animals is frequently associated with inadequate diet, irregular feeding, water deprivation, overcrowding, parasitism, weather extremes, pregnancy, parturition, intercurrent diseases etc. The calving complications which may predispose the disease include abortion or early termination of pregnancy, retained placenta, endometritis and post-parturient metabolic conditions.

Transmission : Ingestion of feed that have been contaminated by the faeces of infected animals, by drinking water in stagnant ponds and by the carrier animals. In housed animals, transmission is via contaminated feedstuff containing improperly sterilized animal

by-products such as bone and meat meal and fish meal. Casual workers, infected clothing and utensils, transportation trucks and birds may transmit the disease to the farm. Active carrier animals shed Salmonella organisms intermittently and without obvious stress factors. Latent carriers with stress factors are also identified in the transmission of salmonellosis.

Human infection is transmitted via contaminated water, raw milk and meat. Compared to bovines, pigs and poultry are more significant sources of infection in humans (see Chapter 4 and 7).

Antemortem findings :

Peracute septicemic form

1. Occurs most frequently in colostrum deficient animals up to four months of age.
2. Increased temperature 40.4°C - 41.5°C.
3. Depression
4. Diarrhoea and dehydration
5. Death within 24 - 48 hours

Approximately four weeks after the onset of diarrhoea

6. Polyarthritis
7. Meningoencephalitis
8. Necrosis of distal limbs, tails and ear

Acute enteritis

9. Common form in adult cattle in late pregnancy and early postpartum
10. High temperature of 40°C - 41°C
11. Depression and loss of appetite
12. Watery, foul smelling diarrhoea and dehydration
13. Emaciation
14. Reduced milk production and abortion
15. Death

Chronic enteritis - Preceded by acute enteric form

16. Further emaciation (poor doer), diarrhoea and dehydration
17. Fluctuating fever (35.5°C - 40.0°C)

Postmortem findings :

Septicemic form

1. Absence of gross lesions in animals
2. Submucosal and subserosal haemorrhage

Acute enteritis

3. Mucoenteritis to diffuse haemorrhagic enteritis
4. Severe necrotic enteritis of ileum and large intestine caused by S. typhimurium
5. Abomasitis in S. dublin infection
6. Enlarged, edematous and haemorrhagic lymph nodes
7. Thickened inflamed gall bladder wall
8. Fatty change of the enlarged liver
9. Subserous and epicardial haemorrhage

Chronic enteritis

10. Areas of necrosis in the wall of caecum and colon
11. Swollen mesenteric lymph nodes and spleen
12. Chronic pneumonia

In the septicemic and acute enteric forms, *Salmonella organisms* are present in the blood, liver, bile, spleen, mesenteric lymph nodes and in intestinal content. In the chronic form, bacteria is present in the intestinal lesions and less frequently in other viscera.

Judgement : Carcass affected with Salmonellosis is *condemned.*

Differential diagnosis : Acute diarrhoea in calves: Diarrhoea caused by infections (such as rotavirus, corona virus, cryptosporidiosis, E. coli), septicemia, dietetic gastroenteritis,

coccidiosis, Clostridium perfringens type C enterotoxaemia

Acute diarrhoea in adult cattle: Bovine viral diarrhoea, coccidiosis, "grain overload", gastrointestinal parasitism, winter dysentery, arsenic and lead poisoning, bracken fern poisoning and intestinal obstruction

Chronic diarrhoea of adult cattle: Johne's disease, copper deficiency and gastrointestinal parasitism

Haemorrhagic septicemia

Haemorrhagic septicemia is a systemic disease of cattle, buffalo, pigs, yaks and camels. It is caused by *Pasteurella multocida* type B of Carter. Outbreaks of this disease are associated with environmental stresses such as wet chilly weather and overworked, exhausted animals. It is specific type of pasteurellosis distinct from of other forms of pasteurella infections.

Transmission : By ingestion of contaminated feedstuff.

Antemortem findings :

1. Disease more severe in buffalo than in cattle
2. High fever up to 42°C
3. Salivation and difficulties in swallowing
4. Cough, and difficult breathing and associated pneumonia in later stages
5. Edematous swelling of throat, dewlap, brisket and peritoneum
6. Diarrhoea

Postmortem findings :

1. Subcutaneous swellings characterized with yellowish gelatinous fluid especially around the throat region, brisket and perineum
2. Enlarged haemorrhagic lymph nodes
3. Haemorrhage in the organs
4. Pneumonia (Fig. 74)
5. Rarely haemorrhagic gastroenteritis
6. Petechial haemorrhage in the serous membranes which are extensive in some cases

Judgement : Carcass of an animal affected with haemorrhagic septicemia is *condemned*. If the disease is diagnosed on antemortem examination, an animal should not be allowed to enter the abattoir. Dressing of such a carcass would create potential danger for the spread of infection to other carcasses.

Differential diagnosis : Anthrax, blackleg, acute leptospirosis, rinderpest, other pasteurellosis, snake bite and lighting stroke.

Fig. 74: Haemorrhagic septicemia. Fibrinous bronchopneumonia.

Calf diphtheria

Calf diphtheria is an acute oral infection of calves less than 3 months old. It is caused by *Fusobacterium (Sphaerophorus) necrophorum*. This agent also causes liver abscesses and "foot rot" in cattle.

Transmission : Fusobacterium necrophorum is an inhabitant of cattle's digestive tract and the environment. Under unhygienic conditions, infection may be spread on feeding troughs and dirty milk pails. Some of the contributory factors for occurrence of this disease include abrasions in the oral mucosa, animals suffering from poor nutrition and other (intercurrent) disease present in young calves.

Antemortem findings :

1. High temperature
2. Coughing
3. Loss of appetite and depression
4. Difficult breathing, chewing and swallowing
5. Swollen pharyngeal region
6. Deep ulcers on the tongue, palate, and inside of cheeks
7. Pneumonia

Postmortem findings :

1. Inflammation and ulceration with large masses of yellow-grey material in the mouth, tongue, pharynx and larynx
2. Often aspiration pneumonia

Judgement : Carcass of an animal affected with local lesions is *approved*. Generalized diphtheric lesions associated with pneumonia or toxaemia require the carcass *condemnation*. The

carcass is also *condemned* if lesions are associated with emaciation.

Differential diagnosis : Vesicular diseases, neoplasms and abscesses

Actinobacillosis

Actinobacillosis is a chronic disease of cattle caused by *Actinobacillus lignieresi*. It is manifested by inflammation of the tongue and less frequently lymph nodes of the head and of even the viscera and carcass.

Antemortem findings :

1. Loss of appetite
2. Salivation and chewing
3. Swollen tongue
4. Mouth erosions
5. Enlarged parotid and retropharyngeal lymph nodes

Postmortem findings :

1. Enlarged tongue showing tough fibrous consistency. ("wooden tongue")(Fig. 75)
2. A cluster of small yellowish nodules and erosions of tongue mucosa
3. Granulomatous lesions in the lymph nodes (Fig. 76)
4. Marked thickening of the lower part of oesophagus and stomach wall
5. Raised plaques and erosions in the mucosa of rumen and reticulum
6. Liver and diaphragm lesions due to contact spread from reticulum

Typical actinobacillosis lesions in the lymph nodes and organs consist of greenish-yellow thick creamy pus with "sulphur granules". These are bacterial colonies surrounded by club like structures

Judgement : Carcass of an animal affected with active progressive inflammatory lesions of actinobacillosis in lymph nodes and lung parenchyma is *condemned*. Condemned material should be sent to authorized rendering plant. If the disease is slight and confined to lymph nodes, the head and tongue and whole carcass are *approved* after the condemnation of lymph nodes. If the tongue is diseased and no lymph nodes are involved the head and carcass are *approved*. The tongue is *condemned*.

Differential diagnosis : Neoplasms, tuberculosis, abscesses in the lymph nodes,foreign body, salivary cysts, fungal granulomas, chronic pneumonia and parasites

Fig. 75: Actinobacillosis. Actinobacillosis of the tongue. The tongue is enlarged, firm and contains numerous granulomatous lesions. It is called "wooden tongue" because of its firmness due to diffuse proliferation of fibrous tissue.

Fig. 76: Actinobacillosis. Multifocal, well demarcated yellow lesions in the retropharyngeal lymph node of a bovine animal.

Actinomycosis ("Lumpy Jaw")

Actinomycosis is a chronic granulomatous disease of cattle and pigs and rarely in sheep and horses. It is caused by *Actinomyces bovis* which is an obligatory parasite in the mucous membrane of the mouth and pharynx. Infection occurs following injury with a sharp object or hard feed pieces to the oral mucosa.

Antemortem findings :

1. Painful swelling of the maxilla and mandible (lumpy jaw); rarely in feet.
2. Suppurative tracts in the granulation tissue breaking towards oral cavity or skin
3. Ulceration of cheeks and gums and wart like granulations outward on head
4. Difficult breathing and salivation

5. Loss of weight
6. Diarrhoea and bloat

Postmortem findings :

1. Lesions in the mandible (Lumpy jaw) or maxilla (Fig. 77)
2. Granulomatous lesions in lower part oesophagus or anterior part of the reticulum
3. Local peritonitis
4. Mild abomasitis and enteritis

Judgement : see Actinobacillosis

Differential diagnosis : Tooth infection, impacted food, bone injury, neoplasms and osteomyelitis due to other causes

Fig. 77: Actinomycosis. Diffuse granulomas in maxilla and formation of green yellow pus. "Sulphur granules" are found in the pus.

Pyelonephritis (Contagious Bovine Pyelonephritis)

Pyelonephritis is a purulent and inflammatory bacterial disease of the kidney pelvis and parenchyma caused by *Corynebacterium renale*. This disease is essentially observed in adult cows and sows. A predisposing factor for developing a kidney infection is trauma to the bladder and urethra during parturition.

Transmission : Infection is spread from clinically normal "carrier cows". The organism enters via vulva from: a) bedding contaminated with urine b) tail swishing by "carrier cows" c) venereal transmission by infected bulls, and d) non sterilized obstetrical instruments.

Antemortem findings :

1. Persistent increased temperature (39.5°C)
2. Loss of appetite and progressive weight loss
3. Painful urination and increased frequency of urination
4. Ammoniac odour from animal
5. Acute abdominal pain (colic)
6. Ceased rumen contraction
7. Decreased milk production

Postmortem findings :

1. Pyelonephritis showing enlarged, pale and greyish coloured kidney (Fig. 78) and enlarged renal lymph nodes. Purulent lesion in the medulla, pelvis and ureters
2. Inflammation of kidney and kidney stones (uroliths, Fig. 79)
3. Enlarged renal lymph nodes
4. Uraemia

Judgement : It depends on infection of one or both kidneys and/or presence of a urine odour. Carcass of an animal affected with

pyelonephritis or nephritis is *condemned* if : 1) renal insufficiency is associated with uraemia; 2) acute infection of the kidney is accompanied with systemic changes in the organs and lymph nodes, and/or degeneration of body tissues. Borderline cases with uraemic odours should be kept in the chiller for 24 hours. They are subjected to a boiling test. If a urinary odour is not present after detention, the carcass may be *approved*.

Subacute or chronic kidney infections with no systemic changes allow for a *favourable judgement* of carcass. Only the affected parts are *condemned*. Pyelonephritis associated with kidney stones often has a *favourable judgement* of the carcass.

Differential diagnosis : Enzootic haematuria in certain areas, post-parturient haemoglobinuria, reticulitis, peritonitis, cystitis, metritis, leptospirosis, Johne's disease, white spotted kidneys of calves, urinary obstruction, infarcts, neoplasms and hydronephrosis

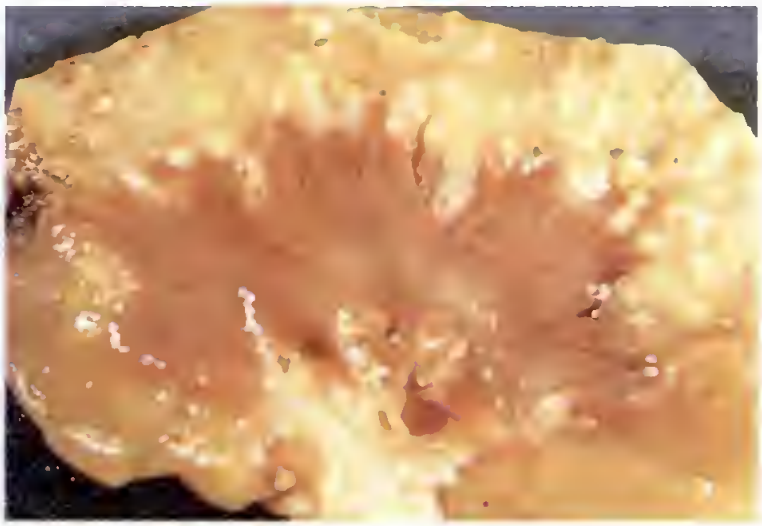


Fig. 78: Pyelonephritis (Contagious Bovine Pyelonephritis). Cut section of kidney showing multifocal abscessation in the cortex and medulla.

Fig. 79: Pyelonephritis associated with urolithiasis (stones). Chemical analysis revealed oxalate composition.

Metritis

Metritis is inflammation of the uterus. This condition is of *bacterial origin*. It occurs as a result of calving problems such as retention of placenta, abortion, twin births, abnormal labour and traumatic lesions of the uterus cervix and vagina.

Antemortem findings :

1. High fever and depression
2. Muscular weakness
3. Placental retention
4. Listlessness
5. Reddish fetid discharge from the vulva

Postmortem findings :

1. Enlarged flaccid uterus showing "paint-brush" haemorrhages on the serosal surface
2. Inflammation of the uterus with light-brown foul smelling

uterine exudate (Fig. 80)

3. Enlarged uterus containing greenish-yellow purulent exudate (Pyometra, Fig. 81)
4. Inflamed peritoneum at the entrance of the pelvic cavity
5. The iliac, lumbar and sacral lymph nodes are enlarged
6. Degeneration of the liver, kidney and heart muscles may be present
7. Congested musculature of the carcass
8. Necrosis of abdominal fat

Judgement : Carcass of an animal affected with acute metritis is *condemned* if it is associated with septicemia or toxaemia. In chronic cases, when toxaemic signs are lacking, the carcass may be *approved* if no antibiotic residues are found.

Differential diagnosis : Recent calving

Fig. 80: Metritis. Necrotizing inflammation of the uterus with greyish-brown foul smelling uterine exudate.

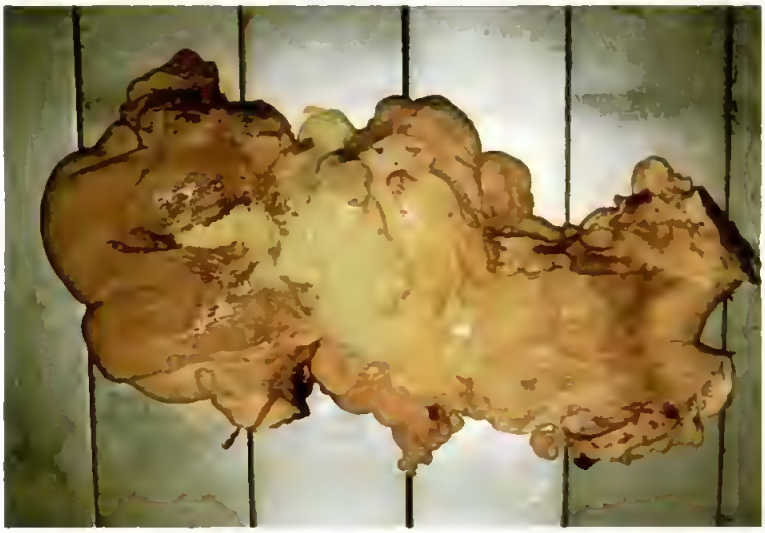


Fig. 81: Pyometra. Enlarged uterus containing greenish -yellow exudate.

Mastitis

Mastitis is inflammation of the udder caused by *bacteria, fungi and yeasts*. Depending on the virulence of the agent and resistance of the udder, mastitis is manifested in acute or chronic forms.

Antemortem findings :

1. Variable temperature depending on stage of condition
2. Swollen warm, painful udder or hard enlargement involving one or all quarters
3. Depression, loss of appetite and dehydration
4. Abnormal gait caused by rubbing of the hind leg against inflamed quarter
5. Animal tends to lie down 6. Purulent or bloody exudate from teats or watery pale fluid in chronic cases

Postmortem findings :

1. Pale yellow granular appearance of the udder parenchyma (Fig. 82)
2. Light brown edematous udder parenchyma (Fig. 83)
3. Enlarged supramammary, iliac and lumbar lymph nodes.
4. Injection sites

Judgement : Carcass and viscera are *condemned* if acute or gangrenous mastitis is associated with systemic changes. If infection has spread from the supramammary lymph nodes via the iliac lymph nodes to the lumbar lymph nodes, this can be taken as evidence of spread of infection from its primary location. The condemnation of the carcass may then be warranted. A localized condition of the udder has a *favourable judgement* of the carcass.

Differential diagnosis : Edema, haematoma and rupture of the suspensory ligament

Fig. 82: Chronic mastitis. Enlarged, firm udder. Incision into the udder parenchyma shows normal milk and pale yellow granular appearance of the udder parenchyma.

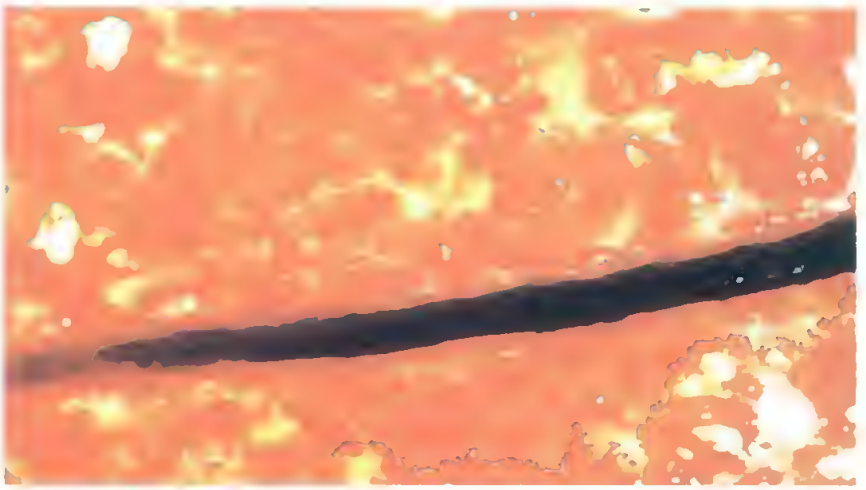


Fig. 83: Brown red edematous udder parenchyma. The udder culture resulted in a heavy growth of Staphylococcus aureus.

Endocarditis

Endocarditis is inflammation of the endocardium of the heart. The lesion is most commonly seen in the valves. It may be the result of *bacteraemia* caused by infection in some remote organs such as the udder, uterus or other sites.

In cattle, the organisms most commonly associated with endocarditis are *Actinomyces pyogenes* and *Streptococcus spp.*. Strains of *Escherichia coli* are also frequently found. The lesion is most commonly found on the valves. Portions of vegetation may become detached and released into the blood stream as emboli which may lodge in other organs. They may be septic or aseptic. The latter contain thrombotic material. Emboli, brought from the right heart to the lungs by blood vessels may cause pulmonary abscesses, or pulmonary thrombosis and the emboli brought from the left heart to the spleen and kidneys may cause septic or aseptic

infarcts in these organs. Abscesses in the heart may also be observed.

Antemortem findings :

1. Moderate fever
2. Breathing with accompanied grunt
3. Pallor of mucosae
4. Loss of condition and muscle weakness
5. Temporary fall in milk production in lactating animals
6. Jaundice and death

Postmortem findings :

1. Large cauliflower-like lesions (Fig. 84) in the endocardium
2. Small wart-like and verrucose lesions in the endocardium
3. Embolic lesions in other organs including the lungs, spleen, kidneys etc.

Fig. 84: Endocarditis. Vegetative valvular endocarditis.

Judgement : Carcass of a debilitated animal is *condemned* for verrucose endocarditis if it is associated with lesions in lungs, liver or kidneys. Carcass affected with ulcerative or verrucose

endocarditis with no signs of systemic changes and negative bacteriological result may be *approved after heat treatment* is applied. Endocarditis showing scar tissue is *approved*. The heart is *condemned*.

Differential diagnosis : Pneumonia, pericarditis, pulmonary edema, emphysema, pleuritis, lymphoma, high altitude disease, congenial heart disease, congenital valvular heart cysts or deformities especially in calves.

Traumatic reticuloperitonitis (TRP, hardware disease, traumatic gastritis, traumatic reticulitis)

TRP is caused from the perforation of the reticulum by a metallic foreign body. It is mostly seen in adult dairy cattle and can occur in beef cattle.

Antemortem findings :

1. Sudden drop in milk production
2. Depression, loss of appetite and weight loss
3. Stretched head and neck
4. Reluctance to walk, arched back and tucked up abdomen
5. Scant, hard faeces, rarely covered with mucus.
6. Mild rumen bloat
7. Audible "grunt" in early stages

If mild *septicemia* develops the animal shows:

8. Elevated temperature (39.2°C - 40°C)
9. Increased heart rate

In chronic localized peritonitis, acute signs and pain lessen, temperature falls and stomach reticulo-rumen motility may return.

Postmortem findings :

1. Adhesions of rumen, reticulum and peritoneum and abscessation
2. Acute or chronic peritonitis
3. Splenic abscessation
4. Traumatic pericarditis (Fig. 85)
5. Metallic objects such as nails, pieces of wire, magnets etc. in the reticulum
6. Lung abscessation or pneumonia
7. Septic pleuritis
8. Edema of the chest

Judgement : Viscera and carcass are *condemned* - a) if the animal is affected with acute diffuse peritonitis or acute infectious pericarditis associated with septicemia; b) carcass with traumatic pericarditis associated with fever, large accumulation of exudate, circulatory disturbances, degenerative changes in organs, or abnormal odour. c) carcass with chronic traumatic reticulo-peritonitis and/or purulent pericarditis with associated pleuritis, abscessation and edema of the chest.

Chronic adhesive localized peritonitis and chronic pericarditis without systemic changes in well nourished animals allow a *favourable judgement* of the carcass. The affected parts of the carcass and organs are *condemned*.

A carcass affected with infectious exudative pericarditis in a subacute stage may be *conditionally approved* pending heat treatment, if bacteriological and antibiotic residue findings are negative.

Differential diagnosis : Uterine or vaginal trauma, abomasal ulceration with perforation, liver abscessation, pyelonephritis, ketosis, abomasal displacement and volvulus, and "grain

overload".

Fig. 85: TRP. Cross section of the heart reveals thick fibrinous deposits that encircled heart. Rusty nail has penetrated through the wall of the reticulum into the pericardium in this case.

Parasitic diseases

Diseases caused by helminths

Lung worms

Dictyocaulus viviparus is a lung worm in cattle causing verminous pneumonia or bronchitis, husk or hoose. Mature lung worms live in the bronchi. During coughing the eggs are swallowed by the host. Hatching of eggs take place in air passages or the digestive tract. Larvae are passed in the faeces. These will survive and develop on the ground if moist and at moderate temperatures they will become invasive in 3 - 7 days. Larvae are resistant to the cold, although their maturation will be delayed.

Upon ingestion by the primary host larvae migrate through the intestinal wall to the mesenteric lymph nodes. From the mesenteric lymph nodes they pass via the lymphatics to the venous circulation and to the heart. From the heart they reach the lung alveoli. Three to six weeks after infection they migrate to bronchi where they mature and lay eggs. They survive 7 weeks in bronchi where they terminate their life cycle.

Antemortem findings :

1. Elevated temperature (40 - 41°C)
2. Rapid shallow breathing which in later stages becomes laboured breathing
3. Nasal discharge
4. Grunting
5. Cyanosis and recumbency.

Post mortem findings :

1. Haemorrhagic inflammation of bronchi with froth
2. Lung edema and emphysema
3. Consolidation of lung parenchyma
4. Lung worms
5. Enlarged lung lymph nodes

Judgement : Carcass of animal affected with lung worms is *approved* if infestation is slight and no secondary changes are observed. The lungs are *condemned*. The carcass is *condemned* if lung worm infestation has caused pneumonia which is accompanied with emaciation or anaemia.

Differential diagnosis : Bacterial bronchopneumonia, abscess, necrobacillosis, tuberculosis, actinobacillosis, hydatid disease and atelectasis

Fascioliasis[3]

Fascioliasis is caused by different liver flukes. *Fasciola hepatica* is the most widespread in distribution. *Fasciola gigantica* in Africa and some parts of South East Asia and *Fasciola magna* found namely in North America including Canada and Europe. In Zimbabwe between 30-70 % of cattle slaughtered are infested with flukes. Usually the liver needs to be trimmed or condemned.

Fasciola hepatica (Fig. 86) is the most common of liver flukes. It is leaf shaped and measures 2.5 cm to 5 cm by 1.3 cm. It lives in the bile ducts of ruminants and other mammals.

Fasciola magna (Fig. 87) is one of the largest of flukes (10 cm by 2.5 cm) noted in the liver and rarely in the lungs of cattle, sheep, deer, moose, elk and other cervidae in Canada. It is found in North America. It may differentiated from Fasciola hepatica by the absence of an anterior cone like projection.

Fasciola gigantica is two or three times larger then Fasciola hepatica. It causes severe economic losses in cattle of Africa.

Fig. 86: Numerous flukes of Fasciola hepatica observed in the bile ducts and liver parenchyma of a cow.

[3] The term "fascioliasis" is commonly used to cover all liver flukes".

Fig. 87: Young fluke of Fascioloides magna taken from a bovine liver.

Life cycle : Each adult is hermaphroditic and produces fertilized eggs which are passed in the bile and faeces onto pasture. The eggs hatch, in the presence of water or moisture into larvae called *miracidia*. If the miracidia find a suitable intermediate host, which is usually the aquatic snail *Limnea truncatula* it will develop into *sporocysts*. In different parts of the world different snails act as intermediate hosts.

The sporocysts divide to form rediae. The *rediae* transform into *cercariae* which are the final larval stage of the cycle. They leave the snail and encyst into a *metacercaria*. After ingestion by a herbivorous animal, the cyst wall is digested in the duodenum and the *larva* crosses the small intestine wall and peritoneal space to the liver. It penetrates the liver and makes its way to the bile ducts and *matures* within a few weeks. The complete cycle of this fluke takes 3 -4 months in favourable conditions.

Antemortem findings :

1. Weight loss and emaciation
2. Fall in milk production
3. Anaemia
4. Chronic diarrhoea

5. Swelling in the mandibular area

Postmortem findings :

1. Emaciated, anaemic or edematous carcass in severe chronic infestations
2. Presence of flukes in enlarged and thickened bile ducts and in the liver parenchyma
3. Hepatic abscesses and secondary bacterial infection
4. Calcification of bile ducts
5. Black parasitic material (excrement) in the liver, lungs, diaphragm and peritoneum 6. Haemorrhagic tracts of migratory immature flukes in the lungs and liver in an acute infestation (Fig. 88)
7. Black lymph nodes of the lungs and liver due to fluke excrement
8. Icterus due to liver damage

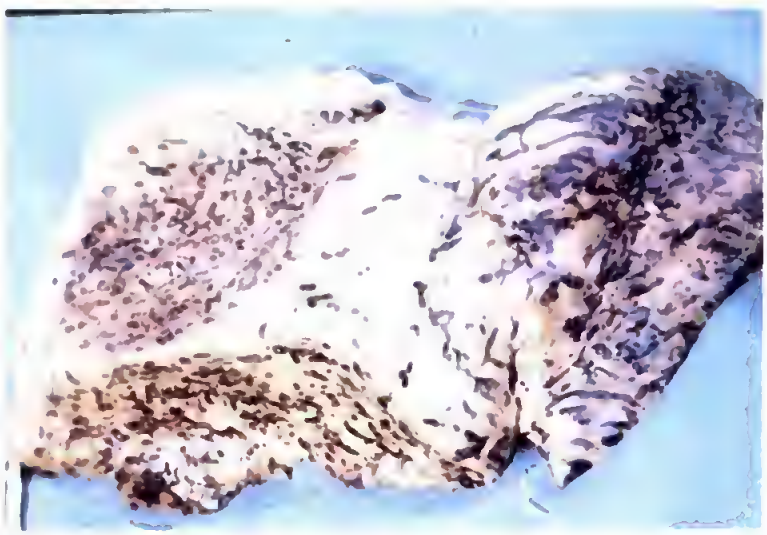


Fig. 88: Acute haemorrhagic tract in the bovine liver.

Judgement : Judgement depends on the extent of the fluke lesions and the condition of the carcass. Severe infestation with associated emaciation or edema would necessitate *total condemnation* of the carcass. Mild, moderate and heavy infestation without emaciation may have a *favourable judgement*. If the parasitic lesions in the

liver are clearly circumscribed, the liver *may be salvaged* after trimming of affected tissue. Otherwise it is *condemned.*

Differential diagnosis : Melanosis, melanoma, Dicrocelium dendriticum and Gigantocotyle explanatum infections in South East Asia

Dicrocoelium dendriticum infection

Dicrocoelium dendriticum (the lancet fluke) is the smallest of the four mentioned flukes in the liver.

Life cycle : Two intermediate hosts are required for its complete cycle. The eggs excreted with faeces by the final host are ingested by a *land snail.* Many species of land snail can act as intermediate hosts where they develop into *sporocysts* and *cercariae.* Cionella lubrica is the principal first intermediate host in North America.

The cercariae are expelled by the snail in mucus and are deposited on plants. They are further ingested by *ants* of the genus *Formica* where they develop into *metacercariae.* Several species of this genus can act as second intermediate hosts. In North America Formica fusca is the second intermediate host.

Ruminants, while grazing, may ingest these ants. The cyst wall of the metacercariae is digested and *larvae* then migrate to the bile ducts where they mature. Dicrocoelium dendriticum is only slightly pathogenic and does not produce clinical symptoms in the animal.

Postmortem findings : In cattle, sheep and swine, the lancet fluke causes moderate thickening of the bile ducts, with slight damage to liver parenchyma. Upon close examination, the parasites can be

seen in the bile ducts.

Judgement and differential diagnosis : see Fascioliasis

Oesophagostomiasis (Pimply gut, Nodular worms)

Oesophagostomiasis is a parasitic disease of ruminants and swine. *Oesophagostomum radiatum* is found in cattle, *Oesophagostomum columbianum* in sheep and *Oesophagostomum dentatum* in swine. The larvae in these species are found in the intestine, caecum and colon. In some Southern African the parasite may affect 5 - 10 % of cattle, sheep and pigs.

Life cycle : The larvae develop to the infective stage on pasture. They are sensitive to cold, dryness and temperature changes. The infected *larvae* penetrate the intestinal mucosa and many of them become encysted. The larvae which penetrate into deeper mucosal layers provoke an inflammatory reaction and nodules of "pimply gut". Further stages of development occur in the intestinal wall. It is believed that many larvae are killed by the reaction they provoke in the intestine. When the larvae leave nodules due to malnutrition or lower resistance of the animal, they reach the colon. In the colon they become *adults* and attach themselves to the colonic mucosa where they lay *eggs*. A great number of nodules disappear as gross lesions after the departure of larvae. With repeated parasitic exposure, the host becomes *immune* and resistant to these larvae and local intestinal reaction becomes granulomatous. The nodules which surround dead larvae and those which calcify after caseation, are persistent and they protrude from the intestinal wall. This may explain why nodules are present in adult animals and why no adult worms are observed in the intestinal lumen. In young animals which have no immunity, adult worms are present in the lumen of the intestine and nodules are

lacking. The are some adults with both, nodules and adult worms in the intestine. O. columbianum in sheep may cause extensive formation of nodules which may become suppurative and may rupture. This further lead to inflammation of the peritoneum and adhesions.

Antemortem findings :

1. Diarrhoea with black-green faeces which may be mixed with mucus and blood
2. Loss of condition and emaciation
3. Stiff gait
4. Young calves may show loss of appetite, diarrhoea, emaciation and anaemia.

Postmortem findings :

1. Greyish white nodules ranging in size from a pinhead to a pea (Fig. 89). The nodules may contain a greenish pasty material in younger lesions or a yellow - brown crumbly material in older lesions.
2. Thickening of the intestinal wall
3. Local peritonitis
4. Mild inflammation of intestine in the acute stage
5. Chronic inflammation of colon in the chronic stage

Judgement : Intestines affected with nodular worms are *condemned*. The carcass is also *condemned*, if severe infestation of this parasite is associated with emaciation and edema. Mild, moderate and heavy infestation without emaciation may have a *favourable judgement*. However, intestines should always be condemned as they cannot be used for sausage manufacture.

Fig. 89: Oesophagostomiasis. Parasitic nodules on the intestinal mucosa (top) and serosa (bottom) in a young bovine animal.

Cysticercosis

Bovine cysticercosis is caused by *Cysticercus bovis*, which is the cystic form of the human tapeworm *Taenia saginata*.

Life cycle : *Cysticercus bovis* is the larval stage of *Taenia saginata*. Taenia saginata may grow from 3 - 7 m in length and lives in the intestine of man. It consists of a suckered head called scolex which is attached to the intestine. It also consist of a neck and hundreds of proglotid segments. Mature proglotids are filled with eggs. The proglotids break off and are excreted in the faeces where they fragment and release the ova. Cattle become infected by grazing on ground and by the digestion of foodstuff contaminated with human faeces. The *oncosphere* liberated in the intestine from the egg penetrates the intestinal wall and through the lymphatics and blood stream reaches the skeletal muscles and

heart. In the muscles the oncosphere develops into the intermediate or *cysticercus* stage containing a scolex. The sites of predilection are the *masseter muscles, tongue, heart* and *diaphragm*. In some countries in Africa the cysticerci appear to show *uniform* distribution in the musculature. If ingested by man, the final or definite host, the scolex attaches itself to the intestinal wall and tapeworms then develop and mature (Fig. 90).

Transmission : Infection in man occurs following consumption of raw or undercooked beef containing viable cisticerci. Cattle become infected by ingestion of feedstuff containing ova passed from infected humans. Cattle raised on free range become often infected through contamination of grazing with human faeces. Infected farm workers may contaminate hay, silage, other feeds or sewage effluent. Intrauterine infection of a bovine fetus was also recorded.

Antemortem findings :

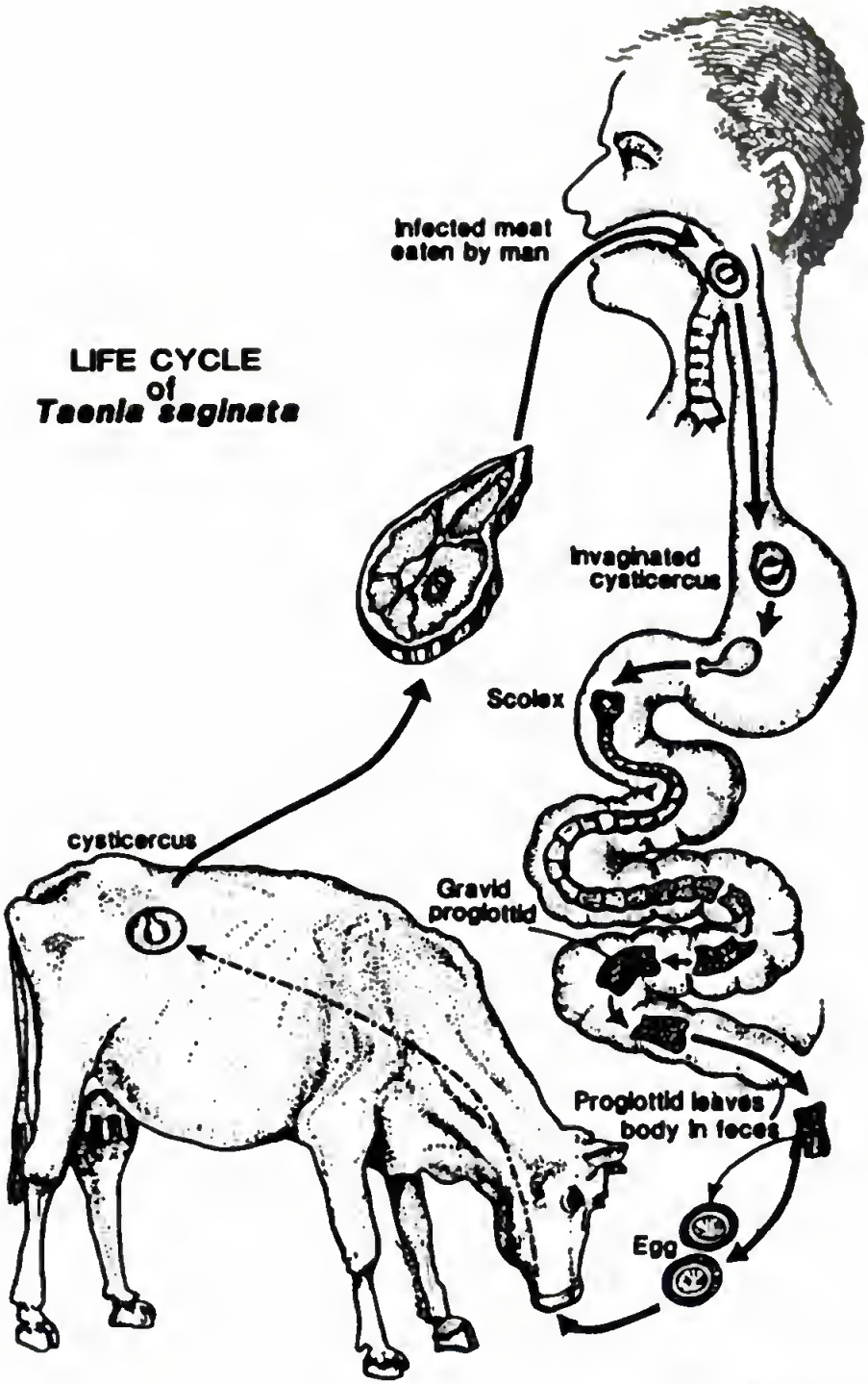
Heavy infestation in cattle may show:

1. Muscle stiffness
2. Rarely fever

Postmortem findings :

1. Small white lesions (cysticerci 2 - 3 weeks after infection) in muscle tissue
2. Clear transparent bladders 5 × 10 mm (infective cysticerci, 12 - 15 weeks after infection, Fig. 91)
3. Opaque and pearl like (over 15 weeks of infection)
4. Degeneration, caseation and calcification (after 12 months or more after infection)
5. Degenerative myocarditis

Fig. 90: Life Cycle of Taenia saginata
(Courtesy of G. J. Jackson, Division of Microbiology,
US FDA, Washington D.C., USA)



Judgement : Carcass and viscera of an infested animal should be differentiated with those with *"heavy" infestation* and those with *"light" infestation*. Carcass and viscera of heavily infested animals are *condemned* and those with light infestation should be treated either by *boiling* or *freezing*. The extent of "heavy infestation" is prescribed by the controlling authority. An animal is commonly considered heavily infected, if lesions are discovered in two of the usual inspection sites including the masseter muscles, tongue, oesophagus, heart, diaphragm or exposed musculature and in two sites during incisions into the shoulder and into the rounds. Generalized infection according to Canadian regulations means 2 or 3 cysts found on each cut into the muscles of mastication, heart, diaphragm and its pillars, and also if 2 or 3 cysts are found in muscles exposed during dressing procedures. In moderate or light infestation consisting of a small number of dead or degenerated cysticerci, the carcass is held depending on the existing country regulations for approximately *10 days at -10°C*.

Differential diagnosis : Hypoderma species (migration to heart), nerve sheath tumour, eosinophilic myositis, abscess and granuloma caused by injections

Fig. 91: Caseous cysticercus. Numerous clear transparent cysts on the heart surface. 0.6 mm in diameter in the heart muscle.

Hydatid disease (Hydatidosis, Echinococcosis)

Hydatid disease in cattle is caused by the larval stages of the 2-7 mm long tape worm *Echinococcus granulosus*, which lives in the intestines of dogs and other carnivores. Several strains of E. granulosus exist, the cattle/dog strain is primarily responsible for hydatid disease in cattle. In Africa hydatid disease is reported more commonly in cattle that are communally owned or are raised on free range, and which associate more intimately with the domestic dogs. Hydatidosis in domestic ruminants inflicts enormous economic damage due to the condemnation of affected organs and lowering of the meat, milk and wool production.

Life cycle : The infective *eggs* containing the *oncosphere* passed in the faeces are accidentally ingested by cattle, sheep, pigs, other animals or humans which act as a *intermediate hosts*. After the infective eggs are ingested by these intermediate hosts, the *oncospheres* in the eggs penetrate the intestine and reach the liver, lungs and other organs including the brain and muscles to develop into *hydatid cysts* at the end of about five months. These cysts measure commonly 5 - 10 cm and contain fluid. Some may reach up to 50 cm in diameter. Others may produce *daughters cysts*. The diagnostic features of a hydatid cysts are a concentrically laminated thick outer layer within which is a germinal layer. In fertile hydatids the germinal layer is granular and has brood capsules each containing protoscoleces. When brood capsules become detached and float free in the cysts fluid they are referred to as hydatid sand. In some animals a fair proportion of hydatid may be sterile. The life cycle is completed when a fertile hydatid cyst is eaten by a *definitive host*, the dog or the appropriate carnivore. Cattle and majority of intermediate hosts show no clinical evidence of infection. However, in *humans* hydatid cysts can cause serious disease.

Antemortem findings : None of significance

Postmortem findings :

Hydatid cysts as described are found in :

1. Liver (Fig. 92), heart (Fig. 93), lungs, spleen, kidneys
2. Muscle and brain
3. Any tissue including bone

Judgement : Carcass showing emaciation, edema and muscular involvement is *condemned* and destroyed. Otherwise the carcass is *approved*. Affected viscera and any other tissue are also *condemned* and destroyed. Burying of carcass is not sufficient, since dogs may retrieve the affected organs.

Differential diagnosis : Retention cysts in kidneys, cysts in liver, granulomatous lesions, Cysticercus tenuicolis and tuberculosis

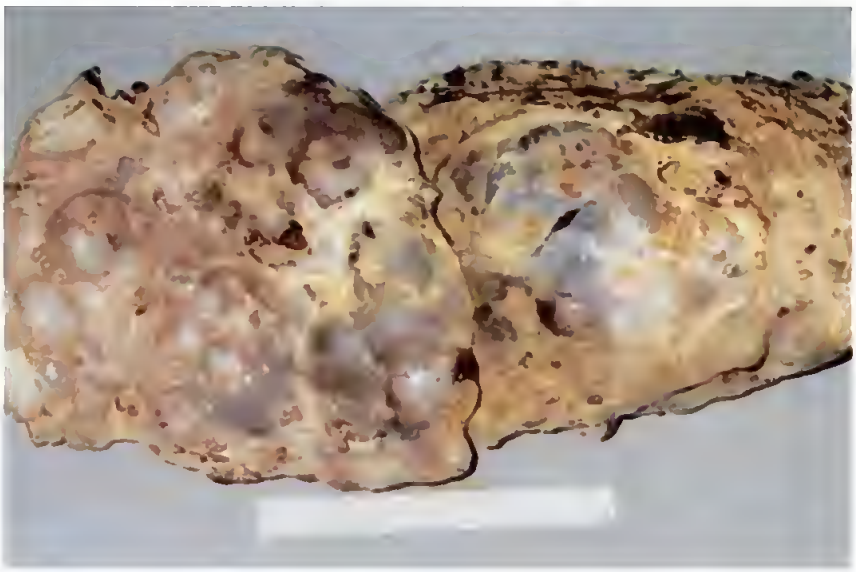


Fig. 92: Hydatid cysts in bovine liver.
(Courtesy Murdoch University, Perth, Australia)

Fig. 93: Hydatid cysts in bovine heart. Note the detached germinal layer.

Onchocercosis

Onchocercosis in cattle is caused nematodes of the genus Onchocerca. Several species are involved, but the most important species is *Onchocerca gibsoni* which causes sub-cutaneous nodules or "worm nests" in cattle in some countries of the Asia-Pacific region and Southern Africa.

Life cycle : The *adult worm* lives in the nodules and the fertilised females liberate *microfilariae* into the tissue lymph spaces from where they are taken up by an insect vector which act as an intermediate host. The common vectors are the midges of the genus *Cullicoides*. Other biting flies can act as intermediate hosts. The *larvae* develop to the infective stage in these insect vectors. Infection of cattle occurs when these biting flies with the infective larvae feed on them.

Antemortem findings : Careful palpation reveals sub-cutaneous nodules in the brisket and buttock regions.

Postmortem findings :

1. Firm fibrous nodules (0.5 cm - 5 cm in diameter) singly or in clusters in the regions of brisket (Fig. 94), buttocks and thighs.
2. The nodules have tightly coiled worms.
3. The worms may be dead or calcified in older nodules.

Judgement : The affected carcasses can be *passed* after the nodules have been *removed*. In heavy infestations the affected briskets are removed, and the tissue and the fascia around the stifle and the brisket are stripped off before the carcasses are passed.

Differential diagnosis : Abscesses, neurofibromatosis, cysticercosis, eosinophilic myositis

Fig. 94: Firm fibrous nodules of Onchocerca gibsoni in the brisket of an ox.

Parafilariasis

Parafilaria bovicola is a filarial parasite of cattle which causes focal cutaneous haemorrhage and sub-cutaneous lesions which are observed as bruising on a dressed carcass. The parasite occurs world wide in countries such as France, Canada, Sweden, South Africa and Zimbabwe, as well as other parts of Africa.

Transmission : The parasite is spread by several haematophagus species of the fly Musca.

Life cycle : During the 7 - 10 months life cycle of the worm, the fly picks up the *egg* off the skin surface of infected cattle. The *larvae* then develop in the fly, and are transmitted to the bovine through the saliva where they migrate subcutaneously and cause the lesions. The lesions appear like a bruise, hence the pseudonym "false bruising". They have a greenish tinge due to the presence of a large number of eosinophils. The *adult filaria* pierces the skin and lays eggs around the periphery of the pierced hole.

Judgement : Lesions vary from mild and localized to severe and extensive. Mild and localized lesions require *trimming* of the affected portions and extensive lesions may warrant a total *condemnation* of the carcass.

Diseases caused by protozoa

Trypanosomiasis

This is a protozoan disease of animals and humans caused by parasites of the genus Trypanosoma, which are found in blood plasma, various body tissues and fluids.

Transmission : Trypanosoma are transmitted primarily by the Glossina spp., tsetse fly, Stomoxys, tabanid and reduviid bugs, and by venereal contact. Trypanosoma species in the insect vector undergo one or two cycles of development.

Antemortem findings :

1. Intermittent fever
2. Anaemia
3. Weight loss and weakness
4. Edema, particulary observed in the face and legs
5. Enlarged body lymph nodes
6. Haemorrhage
7. Opacity of the cornea, keratitis and photophobia

Chronic form of trypanosomiasis is sometimes manifested by progressive weakness, despite absent parasitemia, and death.

Postmortem findings :

1. Enlarged lymph nodes
2. The enlargement of spleen, liver and kidney may also occur.
3. Edematous and emaciated carcass
4. Mild icterus

Judgement : The carcass affected with trypanosomiasis or any other protozoan diseases is *condemned* if an acute condition is associated with systemic body changes. Heat treatment may be recommended in some cases if economically feasible. The carcass of recovered and reactor animals may be *approved* if generalized lesions are lacking. Carcass showing borderline emaciation or slight edema should be examined after the 24 - 48 hours in the chiller. A satisfactory setting would lead to a *favourable judgement* of the carcass. The affected parts of the carcass and organs are *condemned*.

Differential diagnosis : Helminthiasis, malnutrition and other chronic wasting diseases, equine infectious anaemia, heartwater, babesiosis and anaplasmosis

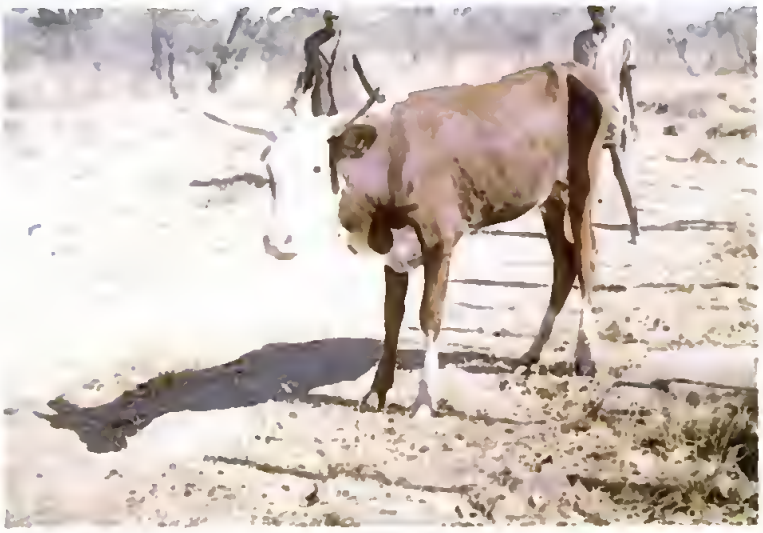


Fig. 95: Trypanosomiasis. This animal shows icteric mucous membranes, weakness in leg muscles and emaciation.

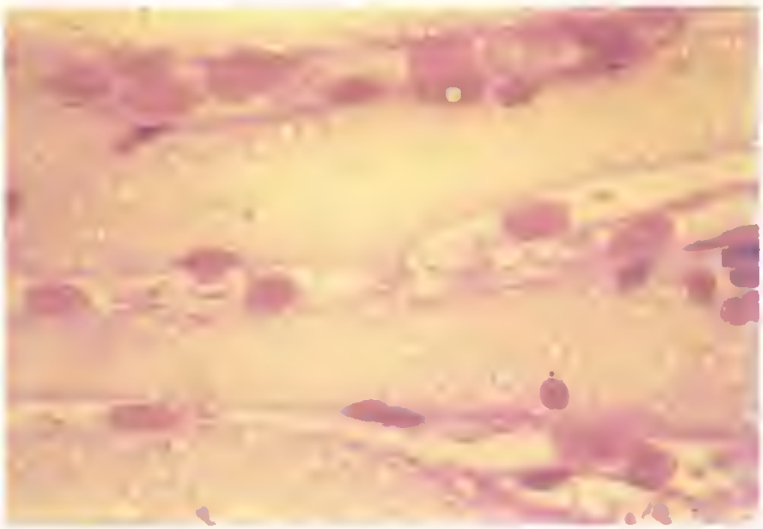


Fig. 96: An impression smear of the trypanosomes and the RBC in the capillaries.

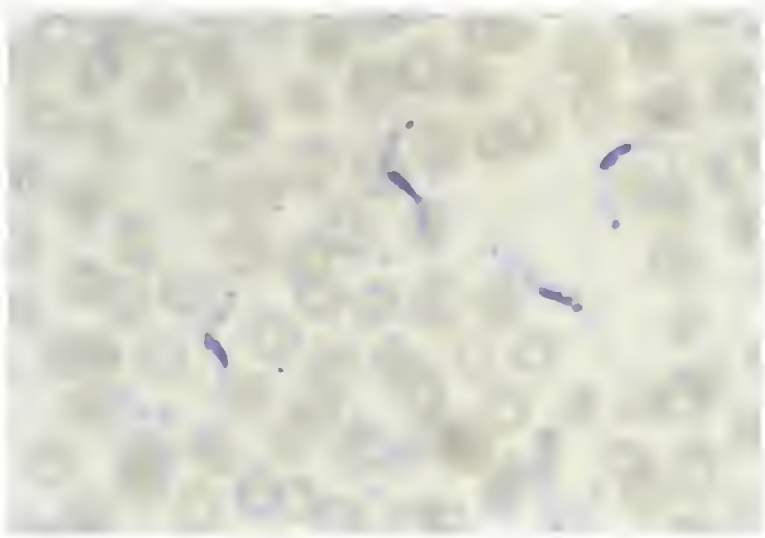


Fig. 97: Trypanosoma vivax in blood smear.

Theileriosis (East cost fever)

East coast fever is a subacute haemoprotozoan disease of cattle caused by *Theileria parva*. Theileriosis is characterized by fever, enlarged lymph nodes, dyspnea and death. In chronic cases loss of condition, emaciation, diarrhoea, blindness, etc. can be seen.

Transmission : Vectors are ixodid ticks of the species *Rhipicephalus*.

Antemortem findings :

1. Mortality up to 90 %
2. High temperature (up to 41°C)
3. Difficult breathing and coughing
4. Nasal discharge, salivation and watery eyes
5. Swelling of the lymph nodes draining the area where the infected tick fed (Fig. 98)
6. Cerebral signs manifested by circling to one side, convulsions and death

Fig. 98: East Coast fever (Theileriosis). Enlarged body lymph nodes.

Postmortem findings :

1. Froth in nostrils and bronchi associated with pulmonary edema and emphysema
2. Swollen, edematous lungs (Fig. 99) and interstitial pneumonia
3. Enlarged and haemorrhagic lymph nodes and splenic lymphoid hypertrophy (Fig. 100)
4. Enlarged and mottled liver
5. Infarcts, thrombosis and lymphoid hypertrophy in spleen (Fig. 100)
6. White spots of lymphoid aggregates in a kidneys
7. Brownish coloration of fat
8. Haemorrhagic and rarely ulcerative enteritis

Confirmation of diagnosis is only made through detection of parasites in a Giemsa stained lymph node biopsy smear and/or blood smear.

Judgement : Carcass and viscera of an animal affected with febrile chronic theileriosis and without systemic lesions are

approved. Carcass is *condemned*, if acute febrile theileriosis is accompanied with fever and generalized lesions. The affected organs are also *condemned*.

Differential diagnosis : Haemorrhagic septicemia, babesiosis, malignant catarrhal fever, trypanosomiasis, Rift Valley fever, heartwater and bovine leucosis

Fig. 99: Theileriosis. Swollen edematous lungs and interstitial pneumonia.

Fig. 100: Theileriosis. Infarcts, thrombosis and lymphoid hyperplasia in spleen.

Besnoitiosis

Besnoitiosis is a chronic debilitating protozoan disease of cattle and horses. It also occurs in wild animals such as antelope, wildebeest (gnu) in Africa and caribou in Canada. The causative agent in cattle is *Besnoitia besnoiti* and *Besnoitia benetti* in horses.

The organism is closely related the genus *Toxoplasma* The mode of transmission is still unknown. It is believed that tabanids are mechanical vectors.

Antemortem findings :

1. Elevated temperature
2. Increased respiration
3. Nasal discharge and lacrimation
4. Diarrhoea
5. Cysts in the skin and subcutaneous tissue and loss of hair
6. Swollen body lymph nodes
7. Severe generalized edema of the head, neck, ventral abdomen and legs
8. Chronic skin lesions show in folding and cracking
9. Decreased milk production
10. Inflammation of the testicles

Postmortem findings :

1. Inflammation of the pharynx, larynx and trachea
2. Sand-like granules and cysts in the turbinates and nostrils (Fig. 101)
3. Sand-like granules in the endothelium of large vessels
4. Dermatitis

Judgement : The carcass is *approved* if the lesions are localized with no systemic involvement. Carcass is *condemned* if

disseminated, generalized lesions are accompanied with emaciation.

Differential diagnosis : Lumpy skin disease, sweating sickness and ectoparasitism (mites, ticks, fungi)

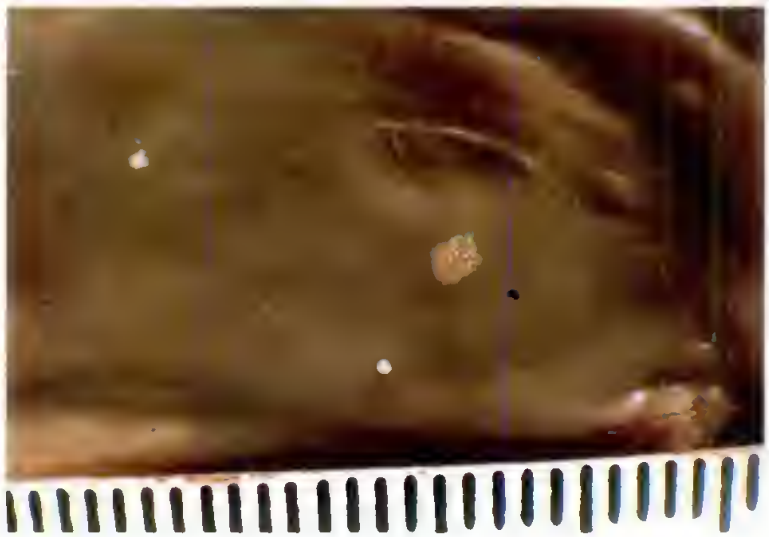


Fig. 101: Besnoitiosis. Sand like granules and cysts in the nostrils of an antelope.

Anaplasmosis (gallsickness)

Anaplasmosis is a rickettsial disease characterized by severe debility, emaciation, anaemia and jaundice and is caused by Anaplasma spp.. They are obligate intracellular parasites. *Anaplasma marginale* is the causative agent in cattle and wild ruminants.

Transmission : Boophilus species of ticks transmit anaplasmosis. Mosquitoes and the horsefly are mechanical transmitters. Transmission is also possible through injection needles.

Antemortem findings :

Acute infection with *A. marginale*

1. High fever
2. Jaundice and anaemia demonstrated by pale mucous membranes
3. Frequent urination and constipation

Chronic infection

4. Emaciation

Postmortem findings :

1. Enlarged and congested spleen (splenomegaly) showing soft pulp
2. Distended gall bladder with dark tarry bile
3. Thin, watery blood, which clots poorly
4. Enlarged, icteric liver, deep orange in colour and distended bile ducts (Fig. 102)
5. Lemon yellow carcass and connective tissue of the sclera of the eye, tendons, pleura, peritoneum, and attachments of diaphragm.

Diagnosis can only be confirmed by detecting parasites in a blood smear stained with Giemsa.

Judgement : Carcass of an animal showing acute infection should be *condemned*. Recovered and "suspect" animals manifesting inconclusive signs of anaplasmosis are *approved* if otherwise healthy. A mildly yellow discoloured carcass may be chilled and assessed after setting. If the discoloration has disappeared, the carcass is *approved* . Animals affected with anaplasmosis could be treated under the supervision of a government official. Guidelines for the withdrawal period for therapeutic agents should be followed if the animals are being shipped for the slaughter.

Differential diagnosis : Icterus and anaemia of different causes, anthrax, leptospirosis, emaciation caused by parasitism and

malignant lymphoma, babesiosis.

Remarks : The access of biting insects to contaminated fresh blood should be prevented. Blood from suspicious carcasses should not be salvaged.

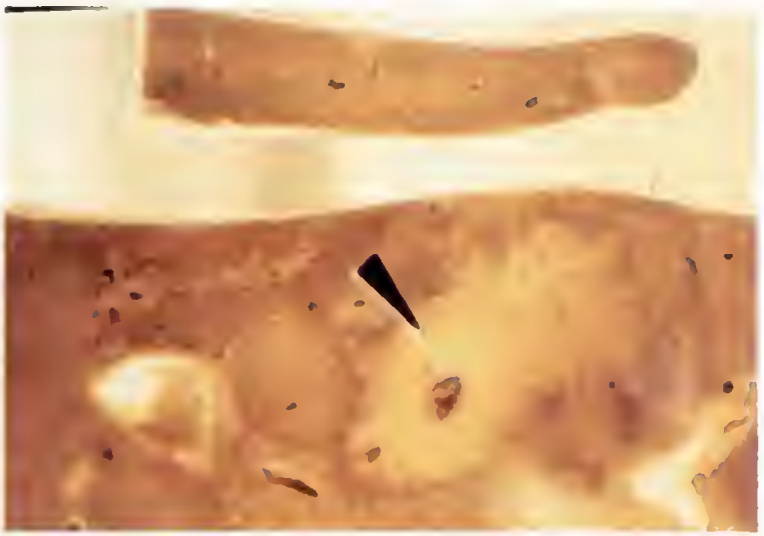


Fig. 102: Anaplasmosis. Ox liver affected with disease showing distended bile ducts.

Babesiosis (Piroplasmosis, Texas fever, Red water fever, Tick fever)

Babesiosis of cattle, horses, sheep and swine is a febrile, tick borne disease caused by various species of the protozoan genus *Babesia*.

Transmission : Different species of ticks in the family Ixodidae serve as vectors in different locations. The Babesia parasites can be transmitted transstadially and transovarially within a tick species.

Antemortem findings :

1. Incubation 7 - 10 days
2. Mortality up to 50 % or over depending on age, breed, etc.
3. High fever (41.5°C)
4. Dark reddish brown urine in the terminal stage
5. Reddened and injected mucous membranes at the early stages and later, anaemic mucous membranes
6. Clinical signs may resemble rabies in cerebral form of babesiosis.

Postmortem findings :

1. Edema and congested lungs
2. Enlarged and yellow liver and distended gall bladder with thick dark green bile.
3. Enlarged spleen
4. Anaemia and pale muscles
5. Jaundice particularly noted in the connective tissue
6. Edematous and haemorrhagic lymph nodes
7. Yellowish-orange colour of musculature (mild cases)
8. Occasionally dark kidneys with no other findings
9. Pink haemorrhage of a bovine brain (Fig. 103)

Diagnosis can only be confirmed by identification of parasite in the peripheral blood smear stained with Giemsa (Fig. 104).

Judgement : Carcass of an animal in acute form of the disease, with associated icterus, is *condemned*. An emaciated, jaundiced carcass showing yellow gelatinous fat also requires total condemnation. A mild form of this disease showing yellow orange coloration of carcass not associated with icterus, may be *approved*. The satisfactory setting of the carcass in the chiller must be considered in this approval.

Differential diagnosis : Anaplasmosis, trypanosomiasis, theileriosis, leptospirosis and bacillary haemoglobinuria.

Fig. 103: Pink haemorrhage. Cerebral form of babesiosis caused by B.bovis. It is characterized by formation of thrombi and emboli in brain capillaries.

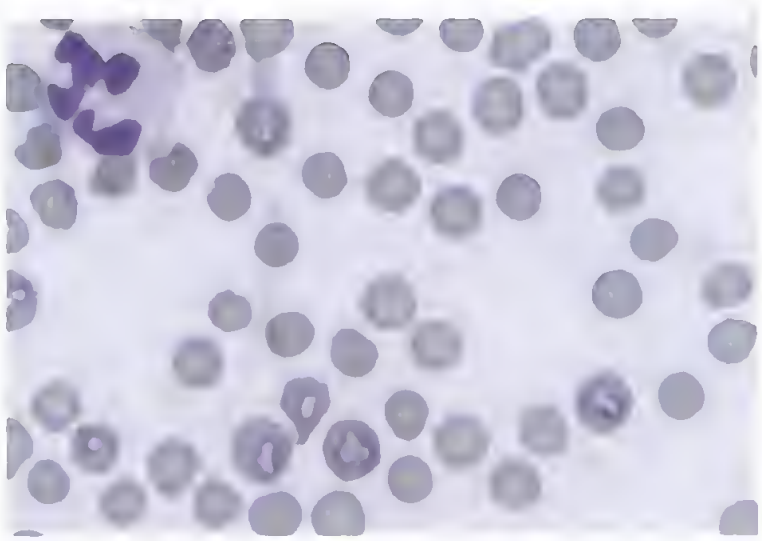


Fig. 104: Babesia bigemina in American bison blood.

Sarcocystosis (Sarcosporidiosis)

Sarcocystosis is caused by the various species of the protozoan genus *Sarcocystis*. This is one of the most common parasitic conditions in domestic food animals and a high percentage of cattle in various parts of the world are infested with these parasites which are usually host specific. In cattle three species have been recognized. They are listed in Table 1. Cattle are the intermediate hosts of Sarcocystis spp. All Sarcocystis spp. in the intermediate hosts, the food animals, are characterised by the formation of cysts in the muscles.

Table 1: Sarcocystis spp. in Cattle

Species	Distribution	Definitive Host/s	Size of cyst	Pathogenicity
S. cruzi	World-wide	Dog, coyote, red fox, racoon and wolf	Microscopic, less than 0.5mm long.	Most pathogenic species in cattle it can cause fever, anaemia, abortion neurologic signs and even death.
S. hirsuta	Probably world-wide	Cat	Macroscopic, up to 8mm long and 1mm wide, fusiform in shape	Mildly pathogenic
S. hominis	Europe	Humans and some primates	Microscopic	Mildly pathogenic to cattle

Life cycle : All Sarcocystis species require two hosts and a pre-predator cycle to complete their life-cycle. A herbivore, the prey, and a carnivore or omnivore, the predator are involved

Sexual development occur in the predator which is therefore a *definitive host*, and asexual development occurs in the prey which is the *intermediate host*.

Two species, one in cattle (S. hominis) and one in pigs (S. suihominis) use humans as definitive hosts and therefore these infections in animals are zoonoses (Fig. 105). Generally speaking dog transmitted Sarcocystis are pathogenic and whereas cat transmitted ones are not.

The most important species in cattle is *S. cruzi* which has world-wide distribution and uses the dog as the definitive host. Sexual development takes place in the dog after which infective sporocysts are passed in the faeces. The details of development in cattle are illustrated in Fig. 106.

The buffalo is the intermediate host for two species: *S. levinei* which forms microscopic cysts and uses the dog as the definitive host, and *S. fusiformis* which forms macroscopic spindle or globular shaped cysts measuring 3.2 cm × 8 mm and uses the cat as the definitive host. S. fusiformis cysts are seen in the oesophagus and the skeletal musculature and is common parasite of the waterbuffalo in many parts of the world.

Transmission : Cattle acquire infection by ingesting sporocysts contaminating feed, pasture or water. After several generations of asexual reproduction by schizogony they form cysts in muscles. S. cruzi, the most pathogenic species for cattle forms microscopic cysts. The definitive host, including humans acquire the infection when they eat bovine tissues containing the viable Sarcocystis cysts. The data provided below are for S. cruzi infestations, unless specified otherwise.

Fig. 105: Life cycle of Sarcocystis hominis (cattle) and Sarcocystis suihominis (pigs) in final host (man) (Courtesy G.J. Jackson, Division of Microbiology, US FDA.Washington D.C., USA)

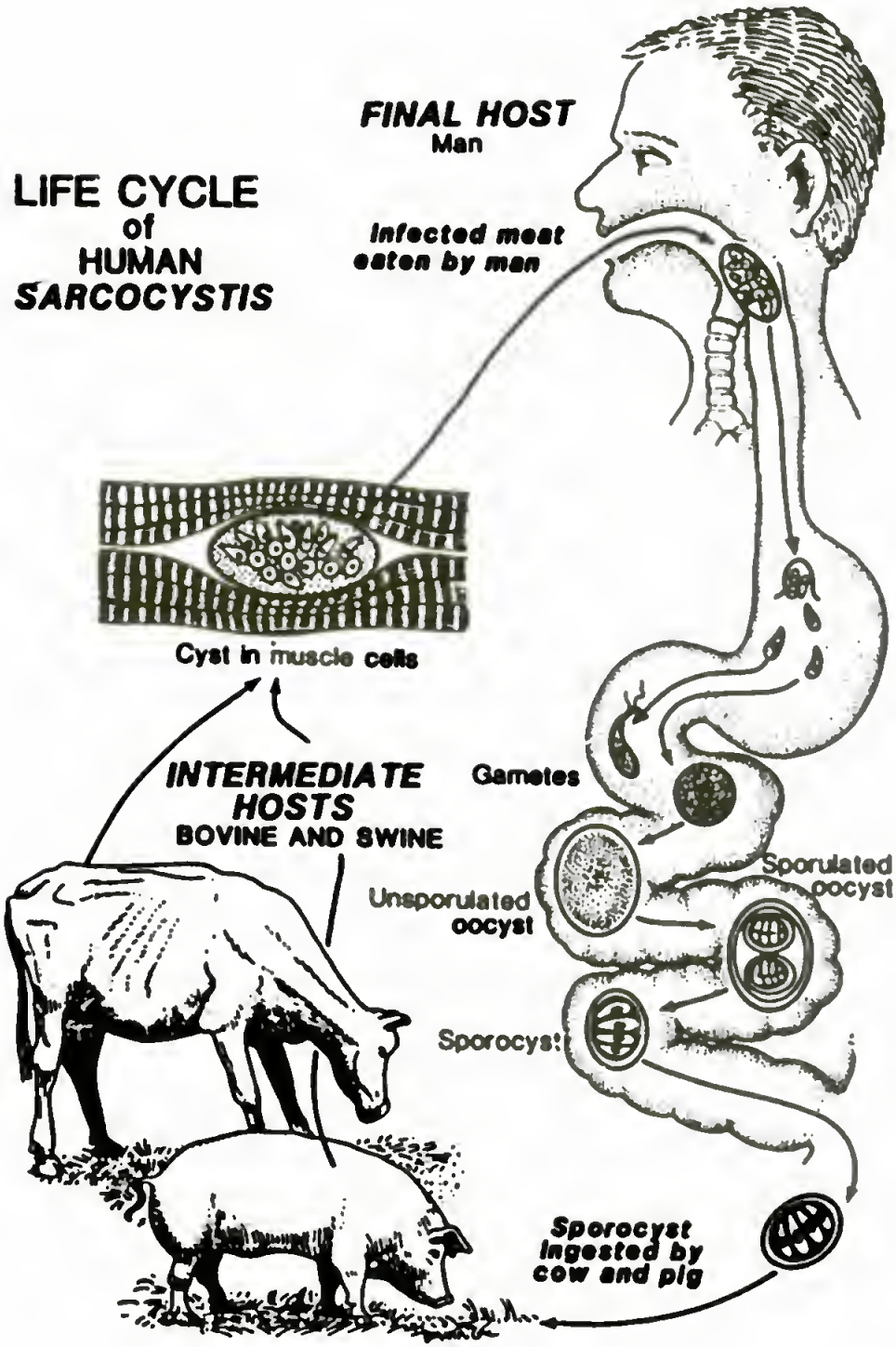
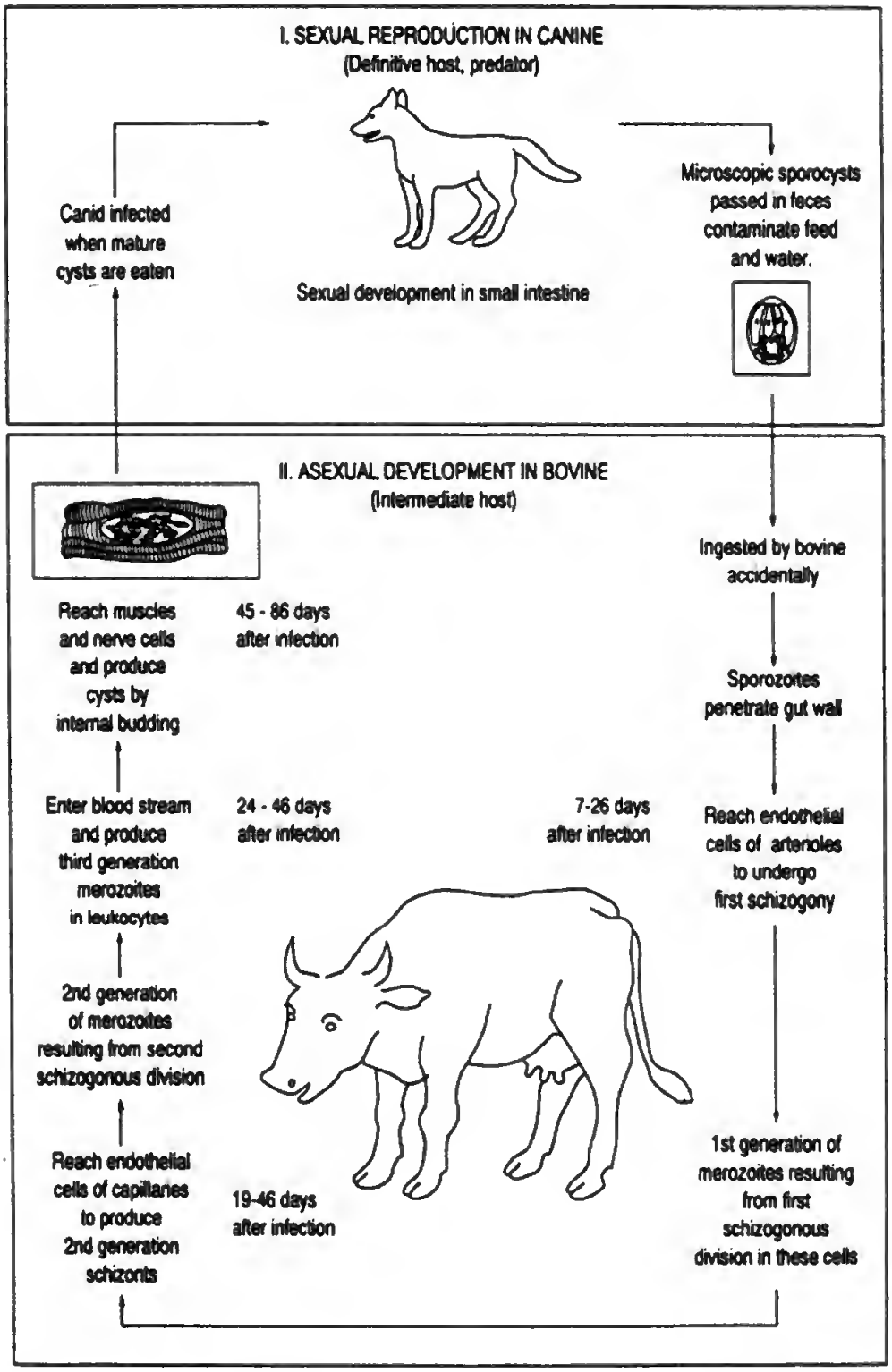


Fig. 106: Life cycle of Sarcocystis cruzi in the bovine and canine (prey-predator cycle)



Antemortem findings :

1. Incubation period 5 - 11 weeks
2. Fever
3. Loss of appetite
4. Excessive salivation
5. Anaemia
6. Abortion
7. Loss of hair especially at the tip of the tail

Postmortem findings :

1. The cysts are microscopic and therefore are not detected on routine postmortem inspection. They cause little tissue reaction.
2. In some cases the cysts may be associated with eosinophilic myositis (Fig. 107).
3. A histological section of bovine muscle affected with eosinophilic myositis showing massive accumulation of eosinophiles and two microcysts of S. cruzi (Fig. 108).
4. S. hirsuta cysts may be seen as fusiform objects 8 mm × 1 mm in the oesophagus, diaphragm and skeletal muscles of older animals especially bulls.
5. Macroscopic cysts of S. fusiformis in the skeletal muscle of buffalo (Fig. 109).

Judgement: Judgement should be made on macroscopic presence of cysts. In heavy and widespread infestations with the visible cysts the whole carcass is *condemned*. In lighter infestations those parts of the carcass which are not affected are *passed* for human consumption. Microscopic examination of muscle may show as much as 70 % infestation in animals worldwide.

Differential diagnosis : Cysticercosis, toxoplasmosis,

neurofibromatosis, eosinophilic myositis

Fig. 107: Sarcocystosis. Eosinophilic myositis.

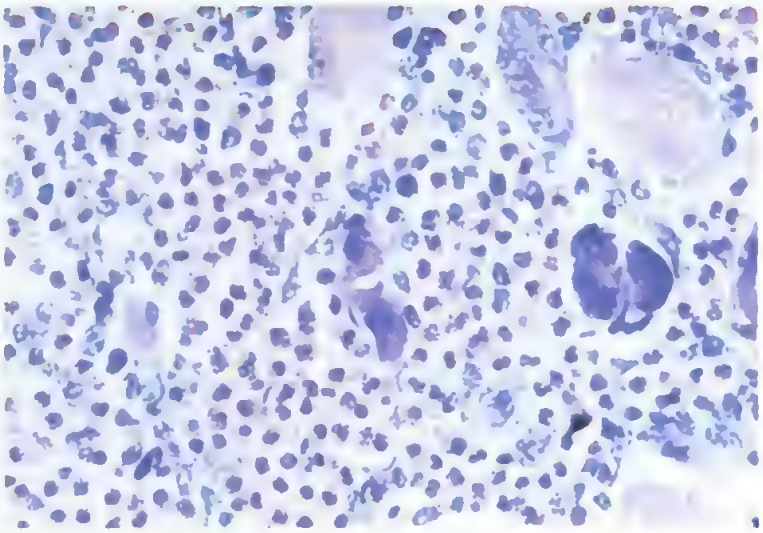


Fig. 108: Histological section showing accumulation of eosinophiles and two microcysts of S. cruzi. There is no tissue reaction.

Fig. 109: S. fusiformis in the skeletal musculature of buffalo.

Diseases caused by arthropod parasites

Hypoderma bovis infestation

There are two warble flies in cattle, Hypoderma bovis and Hypoderma lineatum. They have similar cycles. During the summer the adult fly lays its *eggs* on the leg hair and occasionally on the body of cattle. Within a week the *larvae* hatch and burrow into the skin and, for several months they travel through the body. *Hypoderma bovis* migrates into the thoracic and abdominal cavities towards the spinal canal before moving under the skin of the back. *Hypoderma lineatum* migrates to the oesophageal area before reaching the dorsal area of the animal. In spring (February-May), the larvae reach the area of the back. They burrow a breathing hole and increase in size to approximately 8 mm × 25 mm. They are visible for a month. After this cycle, *maggots* fall to the ground where they develop into *flies* and start the whole cycle once again (Fig. 110).

Antemortem findings :

1. Swelling or eroded skin on the back
2. Larvae protruding from the skin of the back (Fig. 111)
3. Cattle may violently rush and kick the abdomen with hind legs.
4. Erected tail
5. Paralysis of the lower body and legs if the spinal cord is involved.

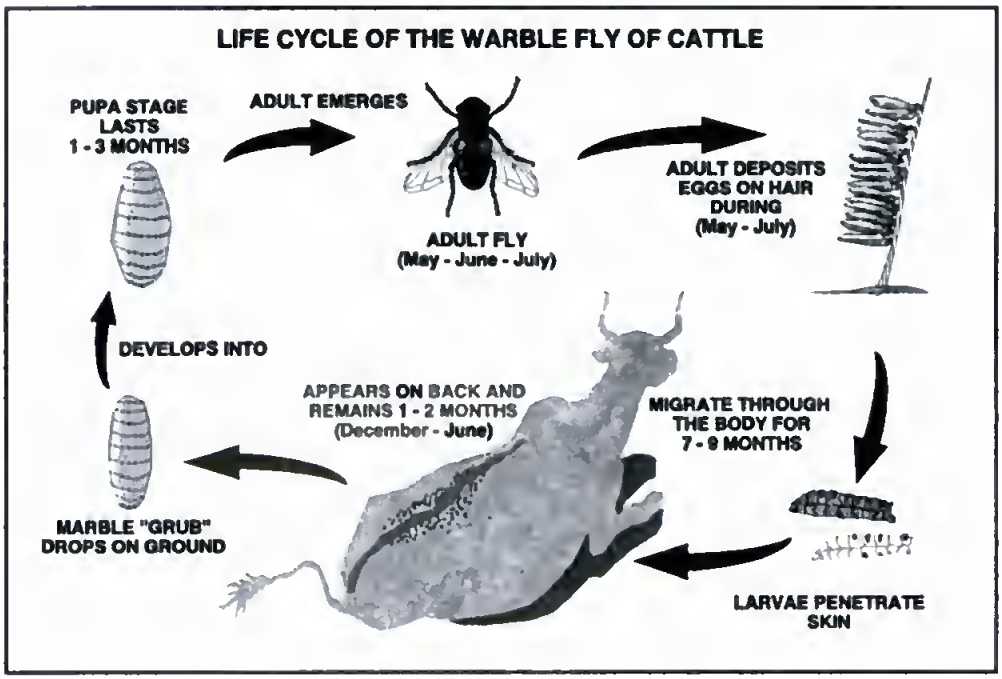
Postmortem findings :

1. Inflamed area of subcutaneous tissue, red, green or yellow in colour, around the maggot or at the site where the maggot lodged
2. Inflammation of the oesophagus may cause rumen bloat due

to obstruction

3. Hypoderma bovis larvae (Fig. 112)

Fig. 110: Hypoderma bovis.
Life cycle of warble fly in cattle



Judgement : Carcass of an animal affected with Hypoderma bovis is *approved*. Subcutaneous lesions are *removed*.

Differential diagnosis : Cysticercus bovis cysts in oesophagus

Fig. 111: Hypoderma bovis. Larvae protruding from the skin of the back in a 2 year old steer.

Fig. 112: Hypoderma bovis larvae.

Screwworm Myiasis

Screwworm Myiasis caused by larvae of the flies *Cochlliomyia hominironux* (New World Screwworm - NWS) and *Chrysomya bezziana* (Old world screwworm - OWS), is characterized by

larvae feeding on living tissues in open wounds of any warm blooded host including humans, resulting in weight loss, other signs of morbidity and sometimes death. The NWS is found in Central and South America including the Caribbean region. The OWS is located in India, Southeast Asia, Tropical Africa and in the Persian Gulf area.

Life Cycle : In the preferred temperature range (20 - 30°C) it is about 21 days. The female, which mates only once, lays one or more batches of up to 300 *eggs* at the edge of any wound or break in the skin in any warm blooded animal. Skin breaks as small as tick bites, as well as natural orifices can be sites of oviposition. The *larvae* develop within 24 hours, and burrow into the living flesh, creating large, deep, open wounds which attract further egg laying females. If unattended, these wounds are often fatal, particularly in newborn animals where the oviposition site is usually the navel.

Antemortem Findings : A serosanguinous discharge often exudes from the infested wounds, and a distinct odour may be detected. In some cases, the openings in the skin may be small with extensive pockets of screwworm larvae beneath. In dogs, screwworm larvae commonly tunnel under the skin. Screwworm infestations in anal, vaginal, and nasal orifices may be difficult to detect, even in the later stages.

Postmortem Findings : After 5 - 7 days of infestation, a wound may be expanded to 3 cm or more in diameter and 5 - 20 cm deep with larvae from a single screwworm egg mass. Usually by this stage, additional screwworm flies have deposited eggs, resulting in a multiple infestation. However, after the death the larvae leave the body due to the temperature reduction and some third stage instar larvae may pupate in the body.

Judgement : The affected carcasses can be *passed* after the wounds tissues have been removed and incinerated.

Differential diagnosis : Other blow flies as *C. Macellaria* and *Sarcophagidae spp.*

Fig. 112A: Wound of adult bovine infested by NWS.

Fig. 112B: Typical pocket like wound from screwworm larvae.

Fig. 112C: Screwworm. Life cycle

CHAPTER 4

SPECIFIC DISEASES OF PIGS

Diseases caused by viruses

African Swine Fever (ASF)

ASF is a highly contagious viral disease of domestic pigs manifested by fever, blotching of skin, haemorrhage of the lymph nodes, internal organs and haemorrhage of the gastrointestinal tract. It is observed in acute and occasionally subacute and chronic forms.

Transmission : There is a natural cycle of the ASF virus between bush pigs, warthogs and giant forest hogs and some tick species (Ornithodorus) in which the virus replicates. The spread of the virus is by contact with affected pigs and infected fomites, ingestion of contaminated uncooked pork garbage, tick bites and contact with domestic and wild carrier pigs.

The virus is quite resistant to cleaning and disinfection. It survives for 2 - 4 months in an infected premises and 5 - 6 months in infected meats. The virus can survive in smoked or partly cooked sausages and other pork products. Humans are not susceptible to this disease.

Antemortem findings :

1. Incubation: 3 - 15 days
2. Fever (up to 42°C)
3. Laboured breathing, coughing
4. Nasal and ocular discharge
5. Loss of appetite and diarrhoea

6. Vomiting
7. Incoordination
8. Cyanosis of the extremities and haemorrhages of skin
9. In chronic stage, emaciation and edematous swelling under the mandible and over leg joints
10. Recumbency

Postmortem findings :

1. Blotchy skin cyanosis and haemorrhage (Fig. 113)
2. Enlarged spleen (splenomegaly, Fig 114)
3. Petechial haemorrhage on the kidneys (Fig. 115)
4. Enlarged and haemorrhagic gastrohepatic and renal lymph nodes
5. Haemorrhage in the heart
6. Hydrothorax, hydropericardium and ascites
7. Haemorrhage of the serous membranes
8. In chronic ASF pericarditis, and emaciated carcass

Judgement : Carcass of an animal affected with African Swine Fever is *condemned*. The animal is prohibited from entering the abattoir.

Differential diagnosis : Hog cholera, salmonellosis, erysipelas, Glasser's disease (Haemophilus suis) infection

Fig. 113: African swine fever. Blotchy skin, cyanosis and haemorrhage.

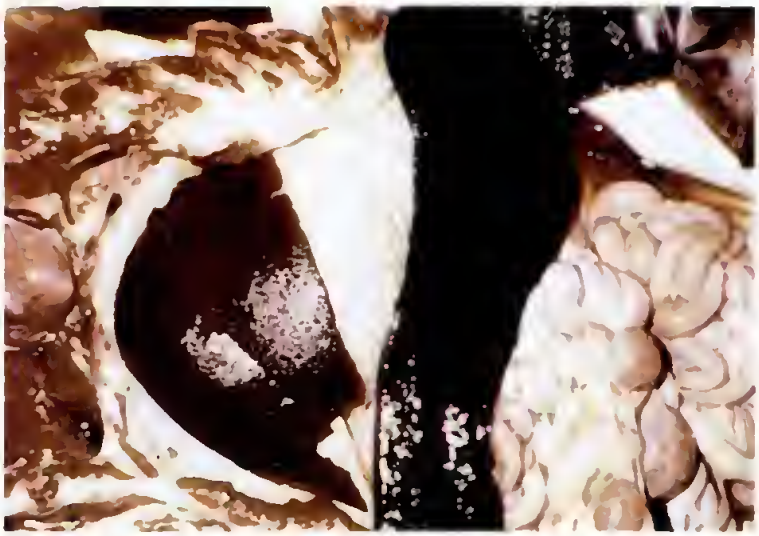


Fig. 114: African swine fever. Enlarged spleen (splenomegaly).

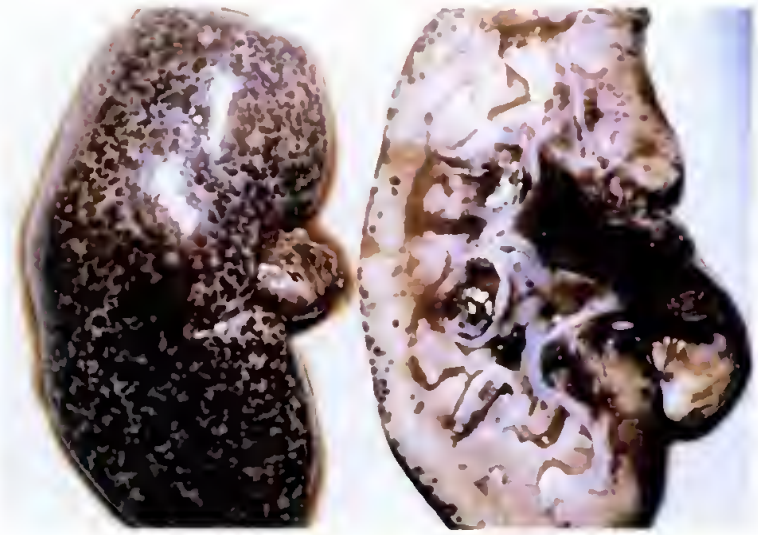


Fig. 115: African swine fever. Petechial and ecchymotic haemorrhage in the kidneys. Note haemorrhagic areas in the renal pelvis and papillae.

Foot and Mouth Disease (FMD, Aphthous fever)

FMD is a contagious, viral disease of swine, cattle, sheep, goats and pigs and other cloven footed animals. The disease in pigs is mild and is important as being a potential danger for transmission to cattle.

Transmission : Direct and indirect contact with infected animals. The virus can also be spread by aerosol, saliva, nasal discharge, blood, urine, faeces, semen, infected animal by-products, swill containing scraps of meat or bones and by biological products, particularly vaccines. Pigs can transmit the disease to cattle and other animals.

Antemortem findings :

1. Incubation 3 - 15 days. Pigs that are fed food wastes contaminated with FMDV may show signs of infection in 1 - 3 days.
2. Snout (Fig. 116) and tongue lesions very common in pigs
3. Dullness and lack of appetite
4. Salivation and drooling
5. Detachment of the skin on a pig's foot (Fig. 117)
6. Shaking of feet and lameness due to leg lesions

Some strains of FMD in swine do not show vesicles but show erosions.

Judgement : Feverish animals with associated secondary bacterial infections call for total *condemnation* of the carcass. The meat of suspect animals may be *conditionally approved* after deboning, and condemnation of the head, feet, viscera and lymph nodes of the carcass. Such meat must be *thoroughly cooked* and could be used as canned meat.

Differential diagnosis : Swine vesicular disease, vesicular stomatitis and vesicular exanthema in pigs can be differentiated from FMD only by laboratory testing.

Fig. 116: FMD. Vesicle on the snout in a pig.

Fig. 117: FMD. Detachment of epithelium on the pig's foot.

Hog cholera

Hog cholera is a highly infectious viral disease of swine manifested by septicemia and generalized haemorrhage. It is noted in acute, subacute and chronic forms.

Transmission : Direct contact with infected pigs and ingestion of uncooked contaminated food wastes containing infected pork scraps.

Antemortem findings :

1. Incubation 5 - 10 days
2. Morbidity 40 - 100 %
3. Mortality 0 - 100 %. Mortality varies with herd susceptibility, virus strain and age of animals.
4. Fever (40.6°C - 41.7°C)
5. Reddened areas of skin
6. Depression
7. Vomiting and constipation
8. Huddling and piling on top of each other
9. Incoordination with staggering gait
10. Tendency to sit like a dog
11. Goose stepping (Fig. 118)
12. Paddling
13. Infection of pregnant cows result in abortion

Postmortem findings :

1. Tonsillar necrosis (Fig. 119)
2. Splenic infarcts (Fig. 120)
3. Button ulcers in the large intestine and intestinal necrosis
4. Haemorrhage of the lymph nodes
5. Pneumonia in chronic infection
6. Petechial haemorrhage in the gall bladder, urinary bladder

and kidneys (Fig. 121); the latter is not present in acute hog cholera.

Judgement : Carcass of an animal affected with hog cholera is *condemned* if kidney lesions are associated with lesions in the lymph nodes and other organs. If the meat appears normal after the organoleptic examination (appearance, taste and consistency), the carcass may be *conditionally approved* pending heat treatment or sterilization. Emergency slaughter of animals affected with hog cholera would require bacteriological examination of the meat in order to eliminate secondary pathogens, mainly Salmonellae. The animals in contact with hog cholera can be *conditionally approved* if heat treatment is carried out.

Differential diagnosis : Erysipelas, septicemic conditions, pneumonia, streptococcosis and salt poisoning

Fig. 118: Hog cholera. Goose stepping.

Fig. 119: Hog cholera. Tonsillar necrosis.

Fig. 120: Hog cholera. Splenic infarcts.

Fig. 121: Hog cholera. Petechial haemorrhage in the kidneys (turkey egg kidney).

Vesicular exanthema of swine (VES)

An acute, contagious, viral disease of swine manifested by the formation of vesicles. Vesicular exanthema is indistinguishable from the other swine diseases such as FMD, VS and SVD.

Transmission : Direct contact with infected animals and ingestion of contaminated uncooked garbage containing infected pork scraps.

Antemortem findings :

1. Incubation: 2 - 4 days
2. Large number of hogs are affected.
3. Heavy mortality in suckling pigs
4. Blotchy rash in unpigmented skin (exanthema)
5. Vesicles on the snout and in the mouth. Ruptured vesicles result in erosions
6. Loss of weight
7. Walking on their knees (Fig. 122) and lameness
8. Squealing when forced to move

Postmortem findings : Vesicles on mucous membranes and skin

Judgement : Carcass of an animal affected with vesicular exanthema is *condemned*, if a feverish animal has generalized lesions throughout the body. In uncomplicated cases when an animal is recovering the carcass is *conditionally approved* pending heat treatment. Laboratory examination should be performed if secondary pathogenic bacteria and/or antibiotic residues are suspected.

Differential diagnosis : Foot and mouth disease, vesicular stomatitis, swine vesicular stomatitis, lameness and leg injuries.

The lesions in the heart and skeletal muscles present in FMD are

not found in vesicular exanthema.

Fig. 122: Vesicular exanthema. Pig walking on knees due to pain from the vesicular lesion on the feet.

Swine vesicular disease (SWD)

A contagious viral disease of swine clinically indistinguishable from vesicular stomatitis, foot and mouth disease and vesicular exanthema of swine.

Transmission : Infected swine, excretions, ingestion of contaminated uncooked pork wastes, minor skin wounds. The shedding of the virus begins before the appearance of clinical signs and may continue for up to 3 months. This virus is more resistant to disinfectants and environmental conditions than the FMD virus. The SVD virus is acid stable and was isolated from certain type of sausage prepared from infected pork meat 400 days after its manufacture. An effective disinfectant for SVD virus is a combination of acid and iodophor disinfectant mixed with detergent.

Antemortem findings :

1. Incubation 2 - 4 days
2. Fever 40 - 41°C
3. Snout (Fig. 123), oral and feet vesicular lesions
4. Lameness

Postmortem findings :

1. Skin lesions
2. Diffuse inflammation of brain on histopathology

Differential diagnosis : Vesicular stomatitis, vesicular exanthema, foot and mouth disease, foot rot, swine pox and chemical and traumatic injuries.

Judgement : Carcass of an animal affected with swine vesicular disease is disposed according to national animal health regulations. In countries with an eradication programme, the carcass is *condemned*. It is also *condemned* in a country free or nearly free of this disease. The animal should not enter the abattoir.

Fig. 123: Swine vesicular disease. Snout vesicular lesion.

Vesicular stomatitis

Viral disease of swine, cattle, horses and occasionally man. The disease is caused by two antigenically distinct types of virus namely Indiana and New Jersey types. Vesicular stomatitis in swine is most commonly manifested by snout and foot lesions.

Transmission : The mode of transmission is not completely known. Biting flies and mosquitoes, direct contact between the animals and droplet infection are possible ways of transmission.

Antemortem findings :

1. Incubation 2 - 4 days
2. Fever
3. Lesion on the tongue (Fig. 124) and snout
4. Lesion in the interdigital space or coronary band
5. Refusal of food but acceptance of water
6. Weight loss
7. Lameness and exungulation

Judgement : Carcass of an animal affected with vesicular stomatitis is *approved*. Affected parts of the carcass and organs are *condemned*. If the disease is not confirmed upon differential diagnosis, the judgement is the same as for swine vesicular disease.

Differential diagnosis : Food and Mouth Disease, vesicular exanthema and swine vesicular disease.

Public health significance : The Indiana and New Jersey viruses are infective for *humans*. Human infection is characterized with chills, fever, malaise and muscle soreness. A mild vesicular stomatitis and tonsillitis may also be present. The recovery of most patients is within a week.

Fig. 124: Vesicular stomatitis. Detachment of epithelium of the pig tongue.

Transmissible gastroenteritis (TGE)

TGE is a highly infectious viral disease of pigs characterized with vomiting, dehydration, diarrhoea and high mortality in pigs up to 3 weeks old.

Transmission : The virus can be spread via aerosol. The virus replicates in the respiratory tract and is excreted in nasal secretion, milk and faeces. Carrier pigs are a major source of infection and transmission of disease. In the herd, the disease spreads from the older pigs to newborn pigs and sows. Suckling piglets get infected by sucking an udder of an infected sow. Uncooked and infected pork scraps may also be the source of infection. Visitors and farm vehicles may transfer the infection to new locations. The virus persists in the infected premises for a few weeks. It can be destroyed with phenol and formalin solution, boiling and drying. Freezing will not destroy the virus.

Antemortem findings :

1. Incubation 24 - 48 hours
2. Transitory fever, but mostly normal temperature
3. Depression
4. Vomiting, pronounced dehydration and profuse diarrhoea
5. Yellow green faeces
6. Death 2nd - 5th day of disease
7. Older pigs may show no clinical signs.
8. Cessation of milk secretion in sows

Postmortem findings :

1. Distended intestine with fluid ingesta (Fig. 125), thin translucent intestinal wall
2. Degeneration of heart muscle and rarely skeletal muscles
3. Microscopical villous atrophy of intestine (Fig. 126)

Fig. 125: TGE. Distended intestine showing translucent intestinal wall and fluid ingesta.

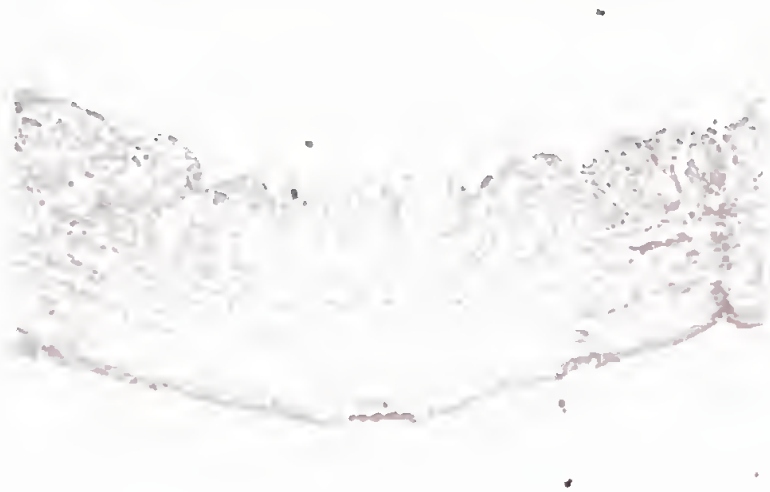


Fig. 126: TGE. Villous atrophy of intestinal mucosa.

Judgement : A slightly affected carcass of an older pig is *approved* if in good condition. The carcass and viscera of an animal showing signs of clinical disease and degeneration of muscle on postmortem inspection are *condemned*.

Differential diagnosis : Hog cholera in early stages of the disease and E. coli enteritis in young pigs. In enteritis, there is no vomiting and it is an enzootic disease.

Diseases caused by bacteria

Pneumonia

Pneumonia is an inflammation of the lungs caused by bacteria, viruses, fungi, parasites or physical or chemical agents. It is frequently accompanied with inflammation of the bronchi, bronchioli and the pleura. Consequently, the term "bronchopneumonia" is commonly used. In pigs, enzootic pneumonia caused by *Mycoplasma hyopneumoniae* and pleuropneumonia caused by *Haemophilus pleuropneumoniae* are most often seen.

Transmission : In infected herds, the infection spreads from the sow to the suckling pigs, and in adult pigs, by common contact and via air. *Mycoplasma hyopneumoniae* is not isolated from the respiratory tract of healthy animals. It persists in chronic lung lesions of recovered animals and is a source of infection particularly for the new animals in the herd.

Actinobacillus (Haemophilus) pleuropneumoniae is found in the nostrils and lungs of healthy animals. An outbreak of the disease may be triggered by environmental stresses.

Antemortem findings :

Enzootic pneumonia:

1. Mortality may occur, but is very low.
2. Fever is usually absent.
3. Acute respiratory distress and a characteristic dry cough when excited

Chronic form:

4. Dry hacking cough
5. Retardation in growth

Pleuropneumonia:

1. Fever (41°C)
2. Respiratory distress
3. Bluish appearance of mucous membranes of the eye and mouth
4. Bloody frothy discharge from nostrils
5. Death

Chronic form

6. Poor feed utilization and emaciation in "carrier" animals

Postmortem findings :

Enzootic pneumonia

1. Plum coloured, greyish consolidation in apical, cardiac and diaphragmatic lung lobes (Fig. 127); lung lobes directed toward the front and bottom are mainly affected.
2. Enlarged edematous bronchial lymph nodes
3. Purulent pneumonia with abscessation usually seen with secondary infection (Fig. 128)
4. Pleurisy and pericarditis may be seen with secondary infection

Pleuropneumonia

1. Bloody froth in wind pipe
2. Generalized consolidation and firmness in the entire lung (Fig. 129)
3. Blood tinged fluid in the chest cavity and abdomen
4. Pus-filled abscesses scattered throughout the lungs.
5. Lesions commonly found in the upper part of lungs and often in diaphragmatic lobe.
6. Whitish clot like adhesions on the lung surface and pleura

Judgement : A carcass affected with *pleuropneumonia* showing healing lesions is *approved*. The affected parts of the carcass and affected organs are *condemned*. *Pneumonia* associated with dry adhesions on the pleura and pericardium and without other lesions may be *conditionally approved*, pending heat treatment. The carcass must be carefully examined for injection sites. If bacteraemia is suspected, bacteriological examination must be performed. Carcass is *condemned* if pneumonia is accompanied with fever and septicemia or emaciation. Pulmonary necrosis with secondary toxic changes in the body also require carcass *condemnation*.

Differential diagnosis : African swine fever, swine influenza,

rhinitis, Ascaris suum infestation, laryngitis, tracheitis, pulmonary edema and congestion, injuries and tuberculosis

Fig. 127: Pneumonia. Enzootic pneumonia. Lung lesions affecting anterior and bottom portions of the lungs.

Fig. 128: Chronic pneumonia with abscessation. This pneumonia was caused by Mycoplasma spp. and later infected with secondary bacteria. A beta-haemolytic Streptococcus was isolated. The animal may also have received antibiotic therapy.

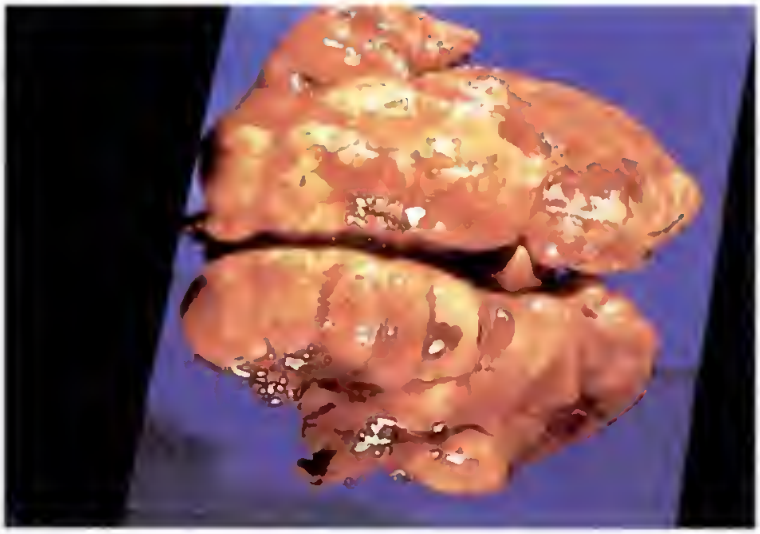


Fig. 129: Porcine pleuropneumonia. Pneumonic lesions throughout the lung tissue. Interstitial emphysema and edema are also noted.

Pleuritis

Pleuritis is the inflammation of the pleura. It is usually associated with pneumonia. An *infectious agent* may reach the pleura by the blood stream, lymphatic system, penetrate from outside the chest cavity, oesophagus or extend from a mediastinal abscess.

Antemortem findings :

1. Fever may or may not be present
2. Shallow, rapid respiration
3. Abducted elbows and unwillingness to move
4. Loss of appetite and weight loss
5. If associated with pneumonia as in pleuropneumonia, a cough may be present

Postmortem findings :

1. Thickening of the pleura and presence of fibrin tags
2. Purulent or fibrinous exudate in the pleural cavity
3. Chronic pleuritis and lung abscessation (Fig. 130)

Judgement : Carcass affected with diffuse fibrinous or serofibrinous inflammation of the pleura is *condemned*. In a case of negative bacteriological and microbial findings, the carcass may be *conditionally approved* with heat treatment. Purulent or gangrenous pleuritis or acute pleuritis associated with inflammation in other organ systems would also require carcass *condemnation*. In localized or chronic pleuritis with no systemic changes, the carcass may be *approved*.

Differential diagnosis : Pneumonia, pulmonary congestion and edema, hydrothorax and haemothorax

Fig. 130: Pleuritis. Chronic pleuritis and lung abscessation.

Valvular endocarditis in pigs

Endocarditis is the inflammation of the endocardium of the heart. Bacterial endocarditis is one of the significant bacterial infections in pigs and other domestic animals.

The etiological agents in swine are *Erysipelothrix rhusiopathiae*, *Actinomyces pyogenes*, *Streptococci spp.* and *Escherichia coli*. Bacteraemia caused by infection in some remote organs, muscle or bones may be associated with a lesion in the endocardium. The valves are the most frequently affected. Emboli may detach from friable vegetation on the valves and pass through the blood stream to organs and cause infarcts. Emboli from the right heart are a frequent cause of pulmonary abscessation or pulmonary thrombosis.

Antemortem findings :

1. Moderate fluctuating fever
2. Loss of condition
3. Pallor of mucosae
4. Arthritis or tenosynovitis

Postmortem findings :

1. Yellow-grey to yellow red valvular lesion (Fig. 131)
2. Embolic lesions in lungs, spleen, kidneys etc.
3. Inflammation of the heart muscle

Judgement : Carcass of an animal affected with endocarditis which had shown fever and loss of condition on antemortem examination and embolic lesions in organs on postmortem examination is *condemned*. Ulcerative or verrucose endocarditis with no signs of systemic changes may be *conditionally approved*, pending heat treatment. The affected organs are *condemned*.

Endocarditis showing infiltration of fibrous tissue is *approved*. The heart is *condemned*.

Differential diagnosis : Pneumonia, pericarditis, pulmonary edema, emphysema, pleuritis, lymphoma, congenital heart disease, congenital valvular heart cysts and deformities

Fig. 131: Valvular endocarditis in a sow heart caused by Streptococcus suis.

Porcine chronic pericarditis

Pericarditis is the inflammation of the pericardium. It is one of the frequent conditions found in swine at slaughter. The agents causing pericarditis include *viruses, mycoplasma, bacteria, fungi* and other microorganisms. It is a common complication of enzootic pneumonia due to secondary invasion with *E. coli*.

Pericarditis can occur secondary to heart infections, systemic infections, or result from metastases of neoplasms arising in remote sites. In swine, fibrinous pericarditis is associated with *hog cholera*, *erysipelas* and *Glasser's disease*. Inflammation of pericardium is also observed in *pasteurellosis, porcine enzootic pneumonia* and *streptococcal infection* in suckling pigs. Primary pericarditis occurs rarely and is of viral origin.

The purulent form of pericarditis is most commonly seen in animals as a result of bacterial, parasitic and mycotic invasions of the pericardium. Direct extension from a surrounding inflammatory area is noted in pneumonia, pleuritis, mediastinal infections, injury to the chest caused by trauma etc..

Antemortem findings :

1. Reluctance to move
2. Shallow respiration
3. Signs of pleurisy and/or pneumonia
4. Signs of heart disorder

Postmortem findings :

1. Deposits of fibrin on the pericardium
2. Pericardium adherent to the epicardium (heart surface) (Fig. 132)
3. Adhesions of pericardium with lungs and/or pleura
4. Purulent inflammation of pericardium

Fig. 132: Porcine chronic pericarditis. Pericardial sac adhered to the heart by thickened fibrous tissue.

Judgement : Carcasses affected with pericarditis should be *condemned* if the inflammation is acute, diffuse or purulent, and associated with fever and systemic changes in other body systems. The rational for condemnation: the pathogenic bacteria are likely to be present in organs and musculature which would present some risk to consumers. A carcass in good condition without systemic changes *can be passed* even though the heart showed evidence of chronic infectious pericarditis. The organs and viscera are *condemned.*

Differential diagnosis : Abscess, Lymphomatosis

Tuberculosis

Tuberculosis is a chronic disease of pigs, manifested by development of tubercles in most organs and is caused by *Mycobacterium bovis* and *Mycobacterium avium.*

The infection occurs primarily by ingestion. The primary complex is incomplete if it develops in the pharyngeal lymph nodes of the head. In such case, the agent entry site is in tonsils. When the bacteria enter through the wall of the intestine, frequently at Peyer's patches, the primary complex includes the mesenteric lymph node. Tuberculosis lesions caused by the bovine type are similar in appearance to those seen in cattle. Avian type lesions in pigs differ from the bovine type.

Antemortem findings : Antemortem findings are similar to bovine tuberculosis. They include:

1. Low grade fever
2. Loss of appetite and emaciation
3. Difficult breathing if lymph nodes of the head, neck, and

lungs are affected.

Postmortem findings :

1. Miliary lesions in the liver (Fig. 133), spleen (Fig. 134) and other organs
2. Lesions in the tonsils, submaxillary, cervical, bronchial, mediastinal and mesenteric lymph nodes.
3. Lesions in the joints and meninges are more common than those in cattle.

M. avium lesions of the lymph nodes are characterized by yellow caseous foci, which range in size from a pinhead to a small pea. Infection from the mesenteric lymph nodes in pigs spreads frequently to the liver by the portal system. Liver infection is common in pigs and is considered localized TB. In infection with M. bovis, tuberculosis liver lesions are part of a generalized infection via the hepatic artery.

Judgement : Carcasses with tuberculosis lesions in the head only are *passed*, after head condemnation. If the carcass contains lesions, it is *condemned.*

Differential diagnosis : Tumours, abscesses and parasitic lesions. The latter are often greenish. Corynebacterium equi infection of the submaxillary lymph node: This lesion can be easily enucleated from its capsule, contrary to tuberculosis lesion which is difficult to enucleate. Lesions caused by Mycobacterium intracellulare and M. scrofulaceum and other acid food organisms are also easily enucleated.

Fig. 133: Tuberculosis. Miliary lesions in the liver tissue.

Fig. 134: Tuberculosis lesions in a pig spleen.

Porcine brucellosis

Brucellosis in pigs is manifested by abortion and sterility in sows, heavy piglet mortality and orchitis in boars. It is caused by *Brucella suis*, although there are also outbreaks of this disease caused by *Br. abortus*. Brucella suis is found more often in adult pigs. It may also affect cattle and horses and is pathogenic for

humans. Meat inspection should carefully examine suspicious livestock and carcasses in order to avoid that cases of brucellosis pass unrecognized in abattoirs.

Antemortem findings :

1. Clinical signs may not be noted.
2. Inflammation and necrosis of testicles in boars
3. Lameness and inco-ordination; may be associated with arthritis and osteomyelitis.
4. Posterior paralysis
5. Abortion and infertility in sows
6. Weak offspring

Postmortem findings :

1. Abscess in the spleen, liver, kidneys, lymph nodes, joint capsule or tendon sheaths
2. Abscess in the testicles or seminal vesicles of boars
3. Catarrhal metritis in sows
4. Arthritis and osteomyelitis of lumbar and sacral vertebral bodies

Judgement : Carcass affected with brucellosis is *condemned*. In some areas *heat treatment* of the carcass may be recommended because of economical reasons. In such cases, the mammary glands, genital organs and related lymph nodes must be *condemned*.

Differential diagnosis : Leptospirosis, erysipelas (if abortion present). Fractures of the lumbar vertebrae with osteomalacia.
In posterior paralysis: Avitaminosis A, deficiency of vitamin B complex factors, and poisoning with rotenone, mercury, organic arsenicals etc.

Porcine salmonellosis

Salmonellosis is one of the most significant infectious diseases of pigs. It is clinically characterized by one of three major syndromes: a *peracute septicemia*, an *acute enteritis* or a *chronic enteritis*. One form of the disease is more prevalent in any particular outbreak. The septicemic syndrome is usually seen in young animals and is generally caused by *Salmonella cholerae suis*. The mortality rate may reach 100 %, with death frequently occurring within a few days. Intercurrent diseases, particularly hog cholera and the nutritional stress caused by a sudden change in diet may predispose to infection with Salmonella organisms such as *Salmonella typhimurium*.

Transmission : Introduction of an infected carrier pig into the herd. Possible infection spread by flies and through farm activities. Healthy pigs may be carriers of Salmonella organisms.

Antemortem findings :

Septicemic syndrome

1. High fever
2. Dark red to purple discoloration of skin especially on the ears and abdomen
3. Nervous signs manifested by incoordination of gait, tremor, paralysis, convulsions, recumbency and death.

Enteric syndrome (mainly seen in adult pigs)

4. Enteritis
5. High fever lasting 12 - 24 hours
6. Severe watery diarrhoea and dehydration
7. Pneumonia
8. Emaciation and death
9. A sequel to enteric salmonellosis may be rectal stricture
10. Abdominal dilatation and frothy to pasty faeces in cases of

rectal stricture

Postmortem findings :

Septicemic syndrome

1. Discoloration of the skin
2. Enlarged and engorged lymph glands
3. Haemorrhages, petechiae and ecchymosis of the epiglottis, stomach, intestine and bladder
4. Enlarged and pulpy spleen

Acute enteric syndrome

5. Necrotic enteritis in the ileum and large intestine with S. typhimurium infection
6. Congestion and hepatization of lungs
7. Marked skin haemorrhage
8. Prominent petechial haemorrhage in the kidneys

Chronic enteritis

1. Areas of necrosis in the wall of the caecum and colon
2. Enlarged mesenteric lymph nodes
3. Chronic pneumonia
4. Abdominal dilatation and low grade peritonitis in cases of rectal stricture

Judgement : Viscera and carcass affected with salmonellosis are *condemned*. In some areas the *heat treatment* of the carcass is recommended because of economical reasons.

Differential diagnosis : Swine erysipelas, hog cholera, swine fever

Swine erysipelas

Swine erysipelas is an infection characterized by diamond shaped skin lesions and in the chronic form, by vegetative endocarditis and arthritis. It is caused by *Erysipelothrix rhusiopathiae*.

Transmission : Healthy carrier pigs shed the bacteria in manure, where they may survive for 5 months. The manure is a reservoir of infection from which bacteria are transferred to non infected piggeries via boots, cloths, birds, flies or other animals. A carrier animal can infect other animals in the same pen.

Antemortem findings:

1. High morbidity
2. Fever in acute stages
3. Conjunctivitis and vomiting in some cases
4. Bright and alert, squealing in pain on movement
5. Pig is lethargic and stops eating
6. Raised red and edematous rhomboid wheals (acute and chronic forms)
7. Sloughing of skin in the area of the rhomboid lesion
8. Swollen joints and lameness (chronic stage)
9. Sudden death in excited animals

Postmortem findings :

1. Arthritis
2. Diamond shaped skin lesions (Fig. 135)
3. Vegetative endocarditis
4. Enlarged and reddened spleen and congestion of the other organs
5. Inflamed and haemorrhagic mucosa of the stomach (paint brush effect) and intestine
6. Cloudy swelling of the kidney and often ecchymotic

haemorrhage

7. Edematous and haemorrhagic lymph nodes

Judgement : An animal affected with an acute disease of erysipelas with erythema or diffuse cutaneous erysipelas with erythema is *condemned* on antemortem inspection because of occupational hazards. Carcass showing skin lesions or arthritis complicated by necrosis or signs of systemic effects is also *condemned*. A localized skin lesion requires only the *removal of skin* and the rest of the carcass is *approved*. Localized endocardial lesions of erysipelas without systemic changes or localized chronic inflammation of joints call for *conditional approval* of the carcass with heat treatment. The carcass may be totally *approved*, if results of a bacteriological examination show that generalized disease is not present, antimicrobial substances are not found and there is no health hazards to consumers and food handlers.

Differential diagnosis : Dermatitis, allergies, external parasites, septicemia, hog cholera, African swine fever, vesicular exanthema, salmonellosis, arthritis and superficial bruises

Fig. 135: Swine erysipelas. Diamond shaped skin lesions.

Melioidosis

Melioidosis is an infectious disease of pigs, goats and occasionally of other animals caused by *Pseudomonas pseudomallei*, present in several tropical and subtropical countries in the Asia-Pacific, Middle East and Caribbean regions. It causes fatal infections in a significant proportion of infected humans, especially those who are immuno-compromised or have intercurrent disease.

Transmission : Infection occurs by ingestion of the infective material containing Pseudomonas pseudomallei, contamination of wounds or abrasions of the skin or perhaps bites of insects. Important sources of infection are rodents, contaminated swamps or muddy water. The organism can live up to three months in shaded soil.

Antemortem findings :

1. Incubation period is variable, longer in pigs than in other animals.
2. Fever up to 41°C especially in sheep and goats
3. Enlarged lymph nodes, particularly the sub-mandibular nodes in pigs.
4. Loss of appetite
5. Posterior paresis and evidence of nervous signs
6. Abortion and still births
7. Orchitis in boars
8. Most cases in pigs are chronic and these may not show clinical signs.

In sheep the clinical signs include high fever (41°C), nasal and ocular discharge and gradual emaciation.

Postmortem findings :

1. Multiple abscesses in most organs especially in the regional lymph nodes, spleen (Fig. 136) and liver
2. The abscesses contain a thick, caseous greenish yellow or off white turgid pus. There is usually no calcification.

In acute cases

3. Pneumonic changes in the lungs
4. Suppurative polyarthritis, the joint capsules contain fluid and large masses of greenish yellow pus.
5. Meningoencephalitis

In sheep the gross finding includes abscesses and suppuration in the nasal mucosae.

Judgement : Carcasses suspected to be infected with melioidosis should be isolated and retained, and the affected tissues, preferably an excised unopened lymph node and the spleen with abscesses should be sent to the appropriate laboratory for examination. Diagnosis is made on the basis of isolation, cultural examination and pathogenicity test. If there are no facilities for retention in isolation, the carcass suspected or tentatively diagnosed to have melioidosis should be *condemned* and properly disposed of. Persons handling the suspect carcass or material should disinfect their hands, forearms, knives and other contaminated equipment. If the laboratory report is negative the carcass has to be treated on *the basis of the lesions present* and the subsequent diagnosis.

Differential diagnosis : Tuberculosis, non-specific purulent conditions. Caseous lymphadenitis in sheep and goats and actinobacillosis in sheep.

Fig. 136: Melioidosis. Multiple abscesses in the spleen of a pig.

Anthrax (see page 80)

Parasitic diseases

Diseases caused by helminths

Trichinosis

Trichinosis has a significant role in food hygiene particularly in countries where meat inspection is poor or inadequate. However, the parasite has not been reported in domestic pigs in a number of countries in developing regions.

Life cycle (Fig. 137): *Trichinella spiralis* is a nematode which parasitizes pigs, dogs, cats, mice, wild boar and other carnivorous game, humans and other mammals. Larvae penetrate the epithelial lining of the small intestine, undergo four moults and become sexually mature adults. The adult worms are 1 - 4 mm long. The newborn larvae pass to the striated muscles by the lymphatics and the blood stream. In the muscle they grow, curl up in a spiral coil

and are encysted (Fig. 138). When they reach the length of 1 mm by 0.5 mm, they appear as oval nodules and are visible to the naked eye. The predilection sites for these larvae are the tongue, diaphragm, eye, masticatory and intercostal muscles. In the muscles, larvae may persist for a long period of time or they may die and become mineralized. Swine, carnivores and humans become infected from eating infected pork, horse, seal or other meat. The digestive juices will cause liberation of larvae from their cysts. They will later develop into adult worms and the cycle will be repeated. Larvae are rarely found in the muscles of cattle, and sheep. Larvae may survive for a long time in decaying and putrefying muscle and carrion.

The most characteristic symptoms of human trichinosis are high fever, weakness, arthralgia, myalgia, abdominal pain with diarrhoea, edema of the face and eyelids, and hives. Neurological symptoms include dizziness and paresis.

Remarks : If the laboratory examination for trichina is not performed in endemic areas heating or cooking and freezing or curing of pork product must be enforced.

Trichinella examination can be carried out as follows:

a) Trichinoscopic examination

Samples are taken from the diaphragm pillar at the transition of the sinewy part. Small pieces the size of an oat kernel are cut. These pieces are then compressed between glass plates and examined by using a microscope or trichinoscope.

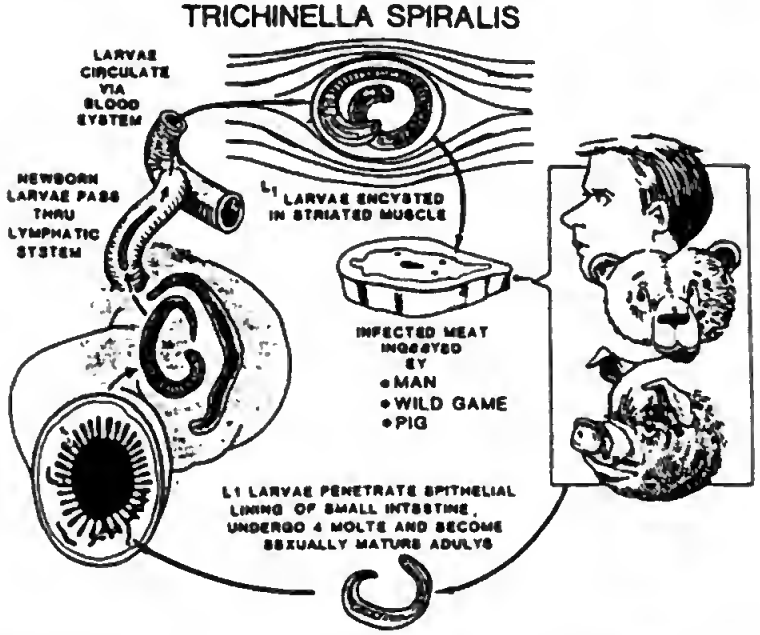
b) Artificial digestion of collective samples

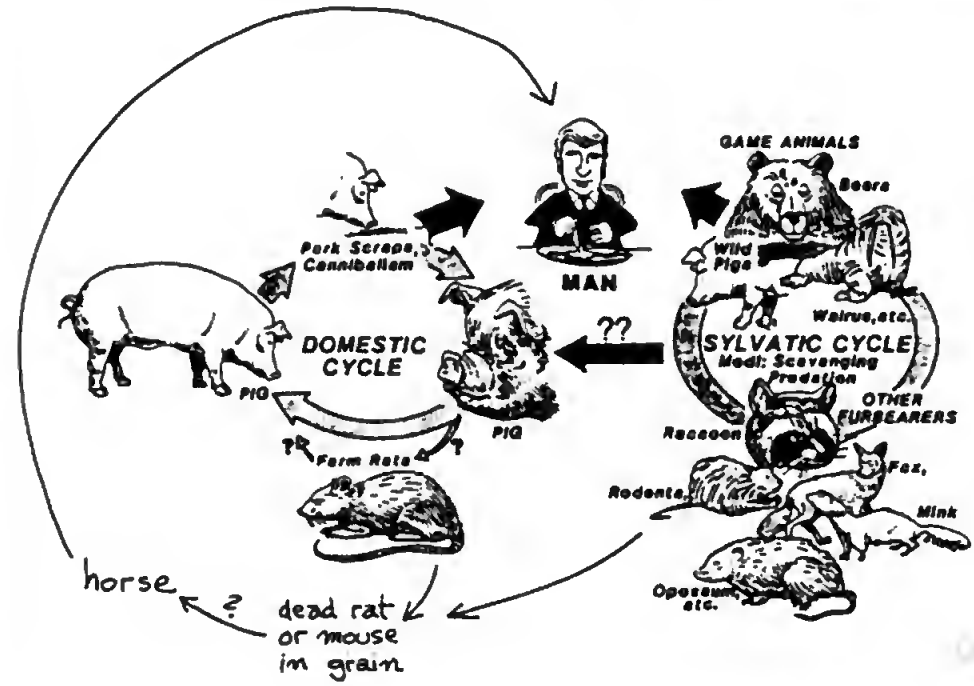
Approximately 1 g is taken from each of a number of carcasses (up to 100), pooled, minced, digested by using a solution of pepsin and hydrochloric acid at 37 -39°C. The fluid in Petri dishes is examined under the microscope at 40 times magnification.

Judgement : Carcass affected with trichinosis is *condemned*.

Differential diagnosis : Sarcosporidiosis and Cysticercus cellulosae infection and tyrosine crystals in muscles

Fig. 137: Life cycle of Trichinella spiralis
(Courtesy G.J. Jackson, Division of
Microbiology, US FDA, Washington D.C., USA)





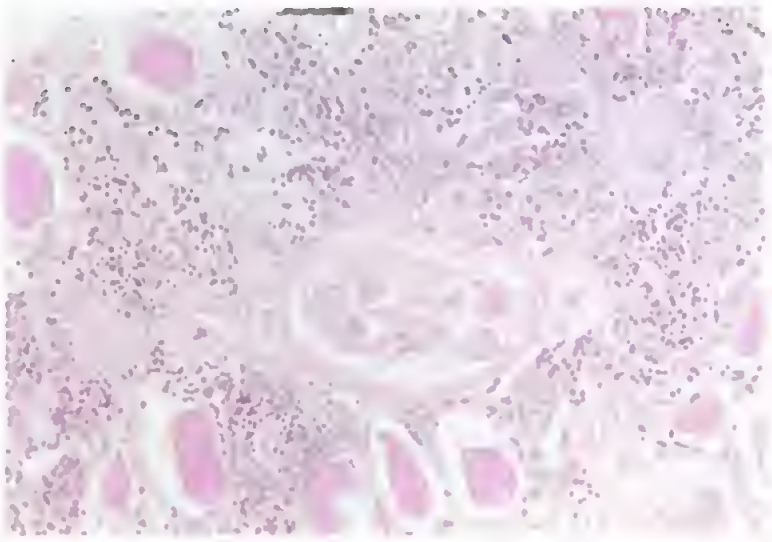


Fig. 138: Trichinella spiralis. Encysted larvae in striated muscles.

Cysticercosis (Cysticercus cellulosae infestation)

Cysticercus cellulosae found in pigs is the intermediate stage of the tapeworm *Taenia solium*, which occurs in the small intestine of humans. T. solium is longer than T. saginata which also occurs in the small intestine of humans.

Cysticerci in pigs are found in the *brain, liver, heart* and *skeletal muscles*. They cause an inflammatory response in the muscle and central nervous system. In humans, auto-infection can occur from the adult worm in the intestine. The most frequently affected site is the central nervous system.

Life cycle : (Fig. 139) After 2 - 3 months of *cysticercus* development in pigs, pearly white *cysts* with an invaginated scolex may be seen in the muscles. With ingestion of infected pork by humans, the *larvae* evaginate and attach to the proximal part of the

live for many years in the environment. The ingestion of proglottides by scavenging pigs is the most frequent way of transmission of cysticerci to swine. *Larvae* hatch from eggs in the pig intestine and they further migrate to muscle tissue, brain, liver and other organs. The use of inadequately treated human excrements as fertilizer is the other cause of porcine cysticercosis. The auto-infection of the central nervous system with the larval form of cysticercus in humans is manifested with headache, dizziness, hydrocephalus, loss of vision and nausea.

Antemortem findings :

1. Fever in acute stages
2. Muscle stiffness

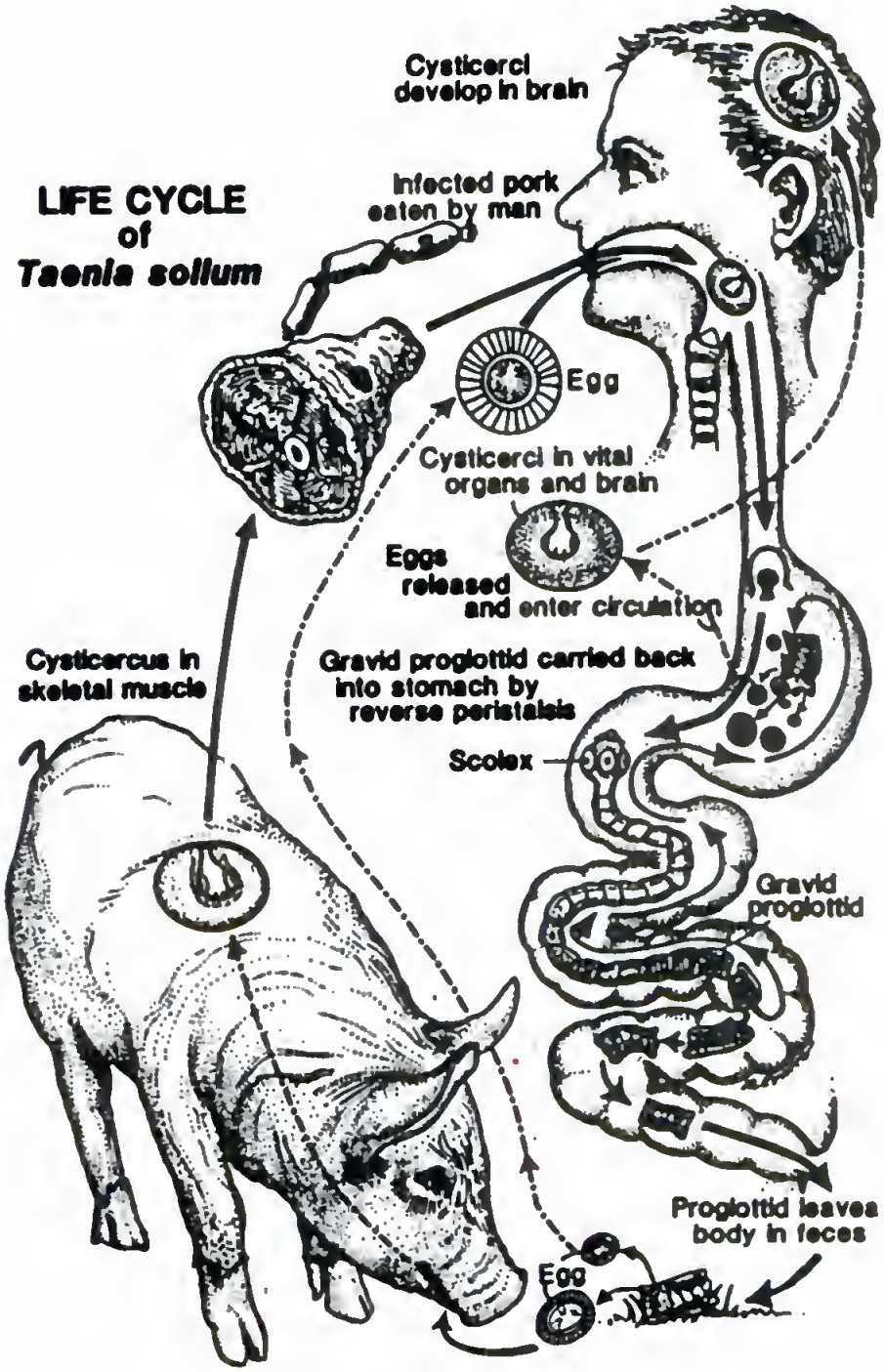
Postmortem findings :

1. Cysts in the heart (Fig. 140) and skeletal muscles
2. Cysts in liver (Fig. 141), brain (Fig. 142) and meninges

Judgement : Heavy infestation with Cysticercus cellulosae calls for carcass *condemnation*. In light or moderate infestation, the carcass may be *conditionally approved* pending heat or freezing treatment. Due to scavenging nature of pigs, infection is usually found only in free range animals and not sty raised ones. Carcasses are usually severely affected ("pearly pork") and are *condemned* despite provision for freezing treatment.

Differential diagnosis : Myositis, abscess and granuloma caused by injection

Fig. 139: Life cycle of Taenia solium
(Courtesy G.J. Jackson, Division of
Microbiology, US FDA, Washington D.C., USA)



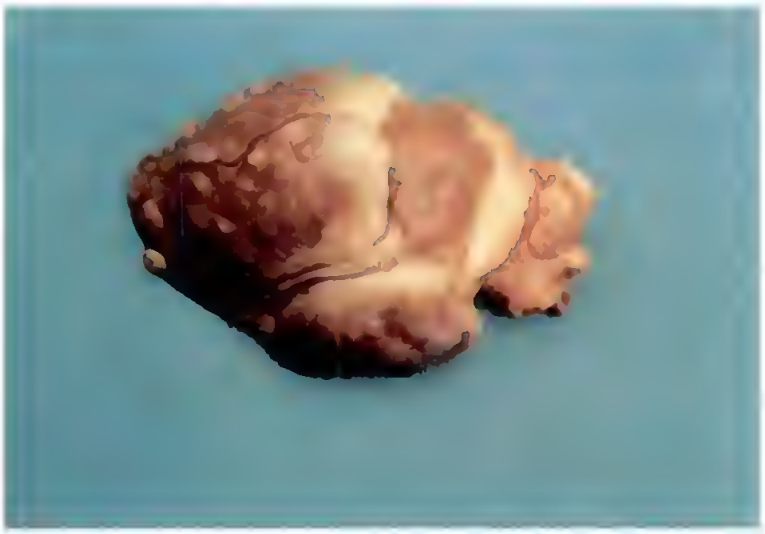


Fig. 140: Numerous cysts of C. cellulosae in the heart muscles.

Fig. 141: Cellulosae cysts in the liver.

Fig. 142: C. cellulosae cysts in the brain.

Ascariasis

Ascaris suum is a pathogenic parasite of mostly young pigs. Ascariasis accounts for significant losses to the swine industry due to reduction in growth rate, stunting of young pigs and liver condemnations. The liver lesions are seen as "milk spots" and degeneration of the liver parenchyma may occur with subsequent cirrhosis. In the lungs, the larvae may cause haemorrhage and frequently verminous pneumonia. Young animals may show marked respiratory signs called "thumps".

Life cycle : *Adult* worms live in the small intestine of pigs where it lays a great number of *eggs*. These eggs have a thick wall, and are resistant to different environments. They may survive in cool, moist surroundings for up to 5 years. The eggs become infective within a few weeks and, if they are ingested by a host, *larvae* are released in the small intestine. The larvae migrate through the intestinal wall and portal vein to the liver within 24 hours of being swallowed. From the liver, larvae enter the blood stream and reach the lungs. The larvae, during this migration, damage the liver and lungs and sometimes the kidneys. Larvae reach the pharynx through the bronchi and trachea, After they are swallowed by the host, they mature in the intestine and lay eggs.

Antemortem findings :

1. Poor growth
2. Rarely cough
3. In severe infections difficult breathing
4. Rarely vomiting up the adult worms

Postmortem findings :

1. Mild inflammation of the intestine and rarely obstruction of the bile ducts caused by adult worms
2. Obstruction of intestine (Fig. 143) by adult worms
3. Large congested liver in early stages
4. Lung edema, haemorrhage or parasitic pneumonia
5. "White spots or milk spots" (Fig. 144) in the liver. These lesions are confluent in chronic cases.
6. Jaundiced carcass and in poor flesh

Judgement : Severely "white spotted" and cirrhotic livers are *condemned*. Mild isolated lesions will disappear if the liver is held overnight in the offal cooler and it can be *released* for human consumption.

Differential diagnosis : Enzootic pneumonia, chronic enteritis caused by Salmonella and Treponema spp.

Fig. 143: Ascariasis. Numerous round worms in the intestine of a market pig.

Fig. 144: Numerous "milk spots" lesions throughout the liver parenchyma.

Sparganosis

Sparganosis in pigs is seen in the Asia-Pacific region and some other parts of the world and is caused by spargana, the larval (plerocercoid) stages of the tape worm *Spirometra erinacei*.

Life cycle : The adult tape worm Spirometra erinacei lives in the small intestine of the cat, fox and dog. The *egg* passed in the faeces develops into a ciliated *coracidium* in water, which when ingested by cyclops (the water flea), the first intermediate host, develops into a *procercoid*. If the cyclops with the procercoid is eaten by a frog, the second intermediate host, the procercoid develops into a *plerocercoid* which resembles the adult tape worm in miniature but without the genitalia. When these frogs are eaten by a cat, fox and dog the plerocercoid develops into *mature tape worms - Spirometra erinacei*. However, if a frog is eaten by a pig or other animals such as snakes the plerocercoid migrates to certain tissues, particularly the skeletal muscles where they appear as cysts up to 6 mm long or as ribbon like structures (Fig. 145)

about 5 cm long with a miniature scolex. These are termed spargana. Humans can be infected with spargana.

Antemortem findings : No significant signs

Postmortem findings :

1. Caseated cysts or cysts up to 6 mm long with spargana in the skeletal muscle or elsewhere
2. Ribbon like structures resembling nerve fibres up to 5 cm long (Fig. 145) in the flank just below peritoneum

Judgement : Carcasses with heavy infestations are *condemned*. In moderate to light infestations the lesions and parasites are removed and the carcass frozen at -12°C or less for 5 or more days before being *passed* for human consumption. Such carcasses may not be acceptable for export.

Differential diagnosis : Sarcocystosis, trichinellosis, cysticercosis, myositis, nerve fibres

Fig. 145: Sparganum. The plerocercoid stage of Spirometra erinacei released from a pig muscle. Note the rudimentary scolex.

Diseases caused by protozoa

Porcine babesiosis (Piroplasmosis, Texas fever, Red water, Tick fever)

Babesiosis of swine, cattle, horses, sheep and swine is a protozoan disease caused by various species of protozoa in the genus Babesia. Babesiosis in swine is caused by *B. trautmani* and *B. perroncitoi*. The percentage of parasitized erythrocytes may be up to 60 % in swine. Pregnant sows may abort. Abortion is associated with febrile animals. It is believed that the source of infection for domestic swine are often feral pigs.

Transmission : Different species of ticks in the family Ixodidae serve as vectors in different locations. Rhipicephalus spp. and Boophilus spp. are vectors in pigs. The Babesia parasites can be transmitted within a tick species. Contaminated surgical instruments and needles may also transmit the infection.

Antemortem findings : Antemortem findings are similar to bovine babesiosis.

1. Fever
2. Anaemia or jaundice
3. Dark reddish-brown urine

Postmortem findings :

1. Fever
2. Thickened bile and reddish-brown urine
3. Congestion of organs
4. Yellowish-orange coloration of the carcass
5. Edematous and haemorrhagic lymph nodes

Judgement : Carcass of an animal showing generalized signs of

infection, jaundice and inadequate bleeding is *condemned*. An emaciated carcass showing yellow gelatinous fat is also condemned. Recovered animals and those showing a mild form of the disease are approved. Satisfactory carcass setting in the chiller is prerequisite for this approval.

Differential diagnosis : Theileriosis, haemobartenellosis, leptospirosis, bacillary haemoglobinuria and eperythrozoonosis, anaplasmosis and trypanosomiasis

Sarcocystosis in pigs (Sarcosporidiosis)

Sarcocystosis of pigs is caused by three species of Sarcocystis. They are: S. miescheriana, S. suihominis, and S. porcifelis. The first two species are macroscopic and when fully developed are fusiform and measure up to 1.5 mm in length. The overall prevalence of sarcocysts in pigs appears to be relatively low and the incidence appears to be decreasing largely due to methods of husbandry where pigs are being reared indoors.

S. suihominis uses humans and non human primates as definitive hosts. It is important as a zoonotic agent. However, it is rarely identified in meat inspection. *S. miescheriana* uses the dog, racoon, wolf and the jackal as definitive hosts. These hosts acquire the infection when they eat the tissues of pigs containing viable cysts of Sarcocystis. S. miescheriana has a world-wide distribution and is pathogenic causing weight loss and purpura. In some countries up to 20 % of carcasses from free range pigs harbour this parasite. *S. porcifelis* has been reported from the former USSR and very little information is available on this species.

Life cycle : The general pattern of life-cycle of Sarcocystis spp. in pigs is similar to that described for S. cruzi in cattle except that

each species of Sarcocystis uses a different definitive host as indicated.

Antemortem findings :

1. Weight loss
2. Purpura of the skin especially of the legs and buttocks
3. Dyspnea
4. Muscle tremors
5. Abortion

Postmortem findings : Elongated fusiform cysts 1.5 × 2 cm in various muscle (Fig. 146)

Judgement : In heavy infestation the carcass is *condemned*. In moderate to light infestations the lesions are *removed* and the carcass *passed*.

Differential diagnosis : Trichinosis, toxoplasmosis, myositis, C. cellulosae

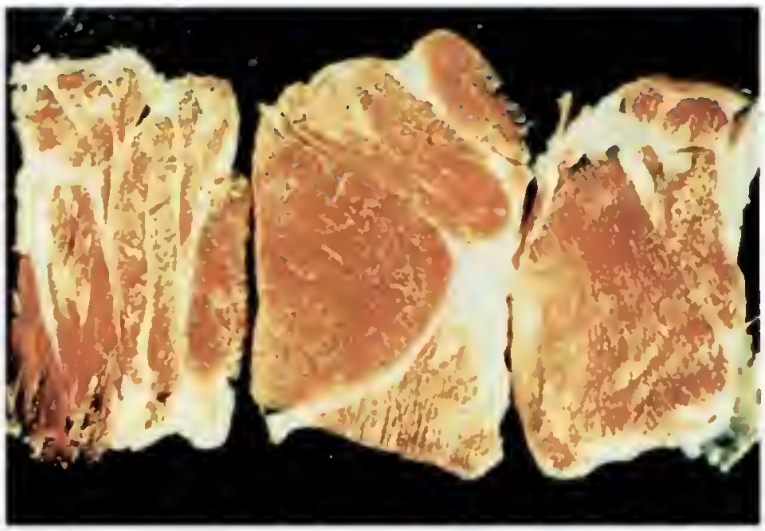


Fig. 146: Sarcocystosis in pigs. Sarcocystis miescheriana in pig muscle.

Toxoplasmosis

Toxoplasmosis is contagious disease of swine, sheep and other species characterized with encephalitis, pneumonia and neonatal mortality. It is caused by protozoon *Toxoplasma gondii* in animals and humans. Toxoplasma is most frequently found in pigs and sheep. Young animals are infected to a lesser degree than old animals. Cattle are rarely affected with clinical toxoplasmosis. Young pigs may die from pneumonia caused by toxoplasmosis.

Humans can get infected with Toxoplasma cysts by ingestion of uncooked animal tissue. In humans clinical symptoms may vary from fever, malaise, skin rash, pneumonia, myocarditis, lymphadenopathy and encephalitis. Infected pregnant women may transfer the tachyzoites to the fetus.

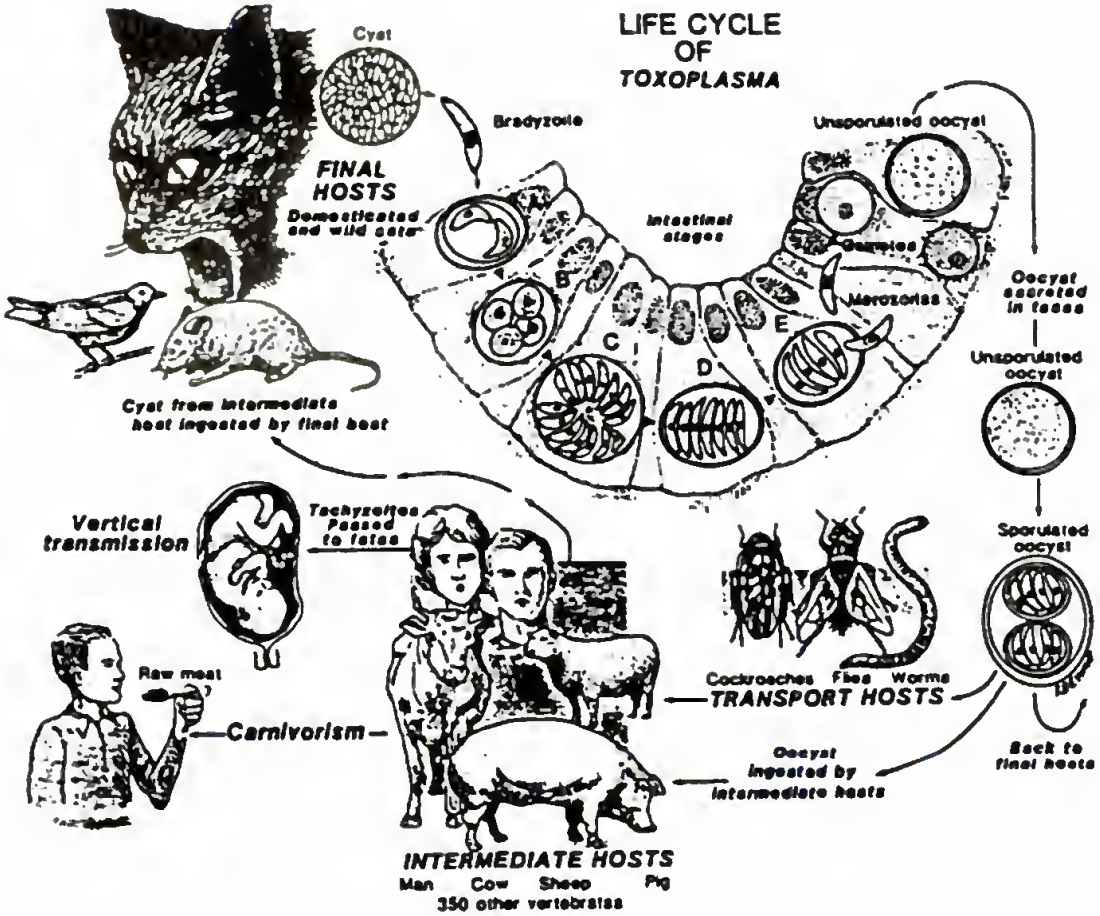
Life cycle : (Fig. 147) Asexual, sexual and oocyst stages of this organism develop in the small intestine of wild and domestic cats. Cats get infected by eating mice or birds or animal tissue containing *infective oocysts*. In the intestine, the parasite develops through the typical coccidian life cycle. *Unsporulated oocysts* are shed in the faeces. After a few days the oocysts sporulate and become infective for over a year. The oocysts are further ingested by the intermediate host (pig, sheep, cattle and humans). From the intestine, oocysts move to various tissues including myocardium, lungs, placenta and most frequently to muscle, brain and liver where they encyst. In the host, they may remain viable for the life span of the host. By eating the infected tissue mice, birds, cats and humans may get infected. The life cycle is then completed.

Antemortem findings :

1. Neonatal mortality
2. Fever (40 - 42°C) and pneumonia in young pigs
3. Difficult breathing and coughing

4. Weakness and wasting
5. Incoordination and trembling
6. Diarrhoea
7. Abortion in pregnant sows and stillbirths

Fig. 147: Life cycle of Toxoplasma
(Coutesy G.J. Jackson, Division of
Microbiology, US FDA, Washington D.C., USA)



Postmortem findings :

1. neumonia
2. Hydrothorax
3. Ascites
4. Intestinal ulceration
5. Necrosis in the liver, spleen and kidneys
6. Inflammation of the lymph nodes
7. Multiple granulomatous lesion in the brain

Judgement : Carcasses of animals showing clinical signs of acute disease are *condemned*. Recovered and reactor animals are *approved*.

Differential diagnosis : *Abortion in pigs*: Brucellosis, leptospirosis, porcine parvovirus infection, hog cholera and Aujeszky's disease. *Encephalitis*: Salt poisoning, chlorinated hydrocarbons, lead, mercury, Vitamin A deficiency, hypoglycaemia, encephalomalacia, meningitis, rabies and scrapie

Miscellaneous conditions

Porcine stress syndrome (PSS)

The susceptibility to PSS is inherited by a single *recessive gene*. This condition is more prevalent in the Pietrain, Poland China, Landrace and Landrace cross breeds of hogs. It is more frequent in short, well muscled meat type hogs. Pork stress syndrome has been commonly associated with the PSE (*Pale Soft Exudative*) condition of the meat. Normal hogs may sometimes produce PSE meat and PSS hogs may produce normal or dry, dark meat. The occurrence of PSE/DFD (*Dry Firm Dark*) meat in pork may be associated with stress particularly during transport, change of

temperature, fighting and chilling. The prevalence of PSE pork is increased in hogs slaughtered in warm weather months as compared to hogs slaughtered in cold months. The pH of PSE pork is less than 6 approximately 1 hour after slaughter and the temperature is above 41 °C immediately after slaughter and drops to 4.4 - 4.6 within 24 hours. In DFD pork the pH usually remains high at about 6 after slaughter or even higher after 24 hours. PSE pork has inferior taste, cooking and cooling qualities.

It is thought that the cause of PSE meat is related to excessive postmortem glycolysis, production of lactic acid, fall in pH with depigmentation and consequently reduced water binding.

Antemortem findings (PSS) :

1. Signs of anxiety
2. Muscle or tail tremor
3. Skin blushing or paleness
4. Mouth breathing
5. Collapse or death

Postmortem findings :

1. Extremely dark, firm, dry pork (Fig. 148)
2. Extremely pale, soft exudative pork (Fig. 148)
3. Visceral congestion and edema

Remarks : Criteria for sensory assessment of PSE/DFD pork include colour and structure.
The following colours are observed:

1. Extremely pale
2. Pale
3. Normal
4. Dark
5. Extremely dark

Muscle may be:

1. Extremely soft, wet
2. Soft
3. Normal
4. Firm, dry
5. Extremely firm, dry

The final assessment of pork muscle can be carried out after chilling of the carcass for 24 hours. The normal setting of meat requires a lowering of pH.

Judgement : Carcass affected with PSE or DFD is *approved* if slight lesions are present. Extensive involvement of the carcass may require *down grading* for manufacturing purposes, or *condemnation*.

Differential diagnosis : Hypocalcemia and pyridoxine deficiency. Both are restricted to home mixed diets. Porcine viral encephalomyelitis should also be considered in differential diagnosis.

Fig. 148: Porcine stress syndrome (PSS). Dark, firm and dry pork (right); pale, soft and exudative pork (left); the normal pork is in the middle.

CHAPTER 5

SPECIFIC DISEASES OF SHEEP AND GOATS

Diseases caused by viruses

Rift valley fever (RVF)

RVF is an acute viral disease of sheep, cattle, goats and humans. It is manifested with hepatitis and high mortality in young lambs and calves, and abortion in adult animals. Rift valley fever resembles influenza in humans. The disease is of significant importance in Africa.

Transmission : Biting insects and mosquitoes. Possible direct contact via cornea. Human infection occur by handling diseased tissues, and strict precautions should be instituted to prevent infection with this virus, such as wearing goggles and gloves.

Antemortem findings :

Sheep

1. Incubation 12 - 48 hours in young animals
2. High morbidity and mortality in lambs and calves
3. Fever
4. Lambs refuse to eat, have abdominal pain and are recumbent.
5. Animals seek a shaded area because of photophobia (squinting and blinking)
6. Photosensitization characterized with a thickened head and ears.
7. Encrustation around the muzzle (Fig. 149)
8. Vomiting in adult animals

9. Congenital malformation of the brain and muscles
10. Abortion in ewes during the illness or convalescence

Cattle

1. Edematous unpigmented skin showing cracking and sloughing due to photosensitization
2. Salivation and inflammation in the mouth
3. Abdominal pain
4. Diarrhoea associated with haemorrhagic inflammation of stomachs and intestine
5. Lameness
6. Cessation of milk production
7. Abortion

Postmortem findings :

1. Cyanotic visible mucosae
2. Necrosis of the liver in lambs (liver may be mottled grey, or reddish-brown to bright yellow in colour)
3. Edematous and haemorrhagic gall bladder
4. Haemorrhage of the gastrointestinal tract, serosae, internal organs and lymph nodes
5. Partial erosions may be seen in the ileum, caecum and colon
6. Udder is purple but inflammation is not observed
7. Haemorrhages in the fetus and haemothorax (Fig. 150)

Judgement : Carcass of an animal showing clinical signs of Rift Valley fever is *condemned*. Reactors and recovered animals are *approved*. Affected parts of the carcass, liver and the blood must be condemned.

Differential diagnosis : Defect in porphyrin metabolism, fungal conditions, acute viremias/toxaemias including enterotoxaemia, bluetongue, bovine ephemeral fever, Wesselbron disease, rinderpest, heartwater, East Coast fever; abortions caused by

Brucella, Vibrio, Trichomonas, Nairobi sheep disease and ovine enzootic abortion

Fig. 149: RVF. Encrustation around the muzzle.

Fig. 150: RVF. Haemorrhages in the fetus and haemothorax.

Contagious ecthyma (contagious pustular dermatitis, orf)

A highly infectious pox virus disease of sheep and goats manifested by the occurrence of the pustular and scabby lesions on the lips, muzzle and udder.

Transmission : Direct contact between animals. Indirect contact with dry scabs in pens. The virus is resistant to drying and may be viable in scabs for months and years in empty feedlots and pens. Farm workers may disseminate the virus among animals of different pens with contaminated equipment, feed and farm vehicles.

Antemortem findings :

1. Incubation: 2 - 3 days
2. Pustular and scabby lesions on the muzzle (Fig. 151), lips and eyes.
3. Lesions on the udder and teats and the coronary band
4. The invasion of lesions by larvae of the screw worm fly and secondary bacterial ection with Fusobacterium necrophorum
5. Lambs and kids are unable to suckle or graze due to lip lesions.
6. Uncomplicated cases may heal within one month.
7. Emaciation
8. Pneumonia in feeder lambs

Postmortem findings :

1. Pustular and scabby lesions on the head, udder and feet
2. Ulcerative lesions in the nasal cavity and erosions in the mucosa of the oesophagus and upper respiratory tract.
3. Inflammation of the reticulum, omasum and intestine
4. Necrotic lesions in the lungs, pleura and liver

Judgement : The carcass is *condemned* if the disease is accompanied with inflammation of the stomachs and intestines, and with bronchopneumonia. Otherwise, it is *approved*.

Differential diagnosis : Bluetongue, sheep and goat pox, ulcerative dermatosis, cutaneous anthrax and vesicular diseases

Fig. 151: Contagious ecthyma. Close up view of a proliferative muzzle lesion.

Bluetongue (BT, catarrhal fever of sheep, "soremuzzle disease")

Bluetongue is a highly contagious viral disease of sheep, manifested by fever, oral lesions, lameness and emaciation. The disease occurs mostly in the African region, but also in Asia and the Pacific and in the Western hemisphere, but can be well controlled by vaccination.

Transmission : Biting insects, especially Culicoides gnats and mosquitoes. Vertical transmission occurs in utero. Semen of infected bulls and mechanical transfer of infected blood by needles.

Antemortem findings :

In sheep:

1. Incubation 6 - 8 days
2. Fever
3. Difficult breathing
4. Excessive salivation
5. Loss of appetite, weakness and emaciation
6. Reluctance to move
7. Mucopurulent to bloody nasal discharge (Fig. 152)
8. Edema of the face, lips and jaw
9. Cyanosis of the tongue and mucous membranes (bluetongue) with erosion and sloughing of the oral mucosa (Fig. 153)
10. Lameness associated with sore feet caused by the inflammation of the coronary band (Fig. 154)
11. Abortion and deformed lambs

In cattle, the disease resembles the infection in sheep and the clinical signs are from unapparent to mild.

Postmortem findings :

1. Vesicles or ulcers in the mouth
2. Generalized edema and haemorrhage of subcutaneous tissue and musculature
3. Excessive mucus in the trachea
4. Congestion of lungs
5. Generalized lymphadenitis
6. Enlarged spleen
7. Necrosis of the heart and skeletal muscles

Judgement : Carcass of an animal affected with bluetongue is *condemned* when the clinical signs of an acute disease are associated with generalized postmortem lesions. The reactor animals are *approved.*

Differential diagnosis :

Sheep: Photosensitization, contagious ecthyma, sheep pox, polyarthritis, footrot, foot abscesses, laminitis, vesicular stomatitis, white muscle disease, muscular dystrophy in lambs, lungworm infestation and pneumonia.

Bovine: Bovine viral diarrhoea, malignant catarrhal fever, infectious bovine rhinotracheitis, stomatitis, laminitis and Ibaraki disease, FMD.

Fig. 152 : Blue tongue. Mucopurulent to bloody nasal discharge.

Fig. 153 : Blue tongue. Intense congestion and swelling of lips and gums and sloughing of the dental pad mucosa.

Fig. 154 : Blue tongue. Close-up view of a lesion on the coronary band of a sheep.

Sheep and goat pox

Sheep and goat pox is a contagious viral disease of sheep and goats manifested by papular and pustular eruptions on the skin and in generalized conditions with haemorrhagic inflammation of the respiratory tract.

Transmission : Direct contact with infected animals, aerosols of nasal secretions and saliva and dried scabs. Indirectly by fomites and transportation vehicles.

Antemortem findings :

1. Incubation 6 - 8 days
2. Fever
3. Laboured breathing
4. Depression
5. Lacrimation and salivation
6. Lesions on the muzzle and lips (Fig. 155)

7. Skin lesions may vary from macules, papules, vesicles, pustules to pocks and scabs.
8. Necrosis and coalescing of the lesions and loss of wool (Fig. 156)
9. Clinical signs of goat pox are less severe than in sheep pox. The benign form of sheep pox is commonly found in adult sheep and the malignant form in lambs.

Postmortem findings :

1. Reddish to whitish firm nodules in the mucosa of the pharynx and trachea
2. Reddish to whitish nodules in the lungs (Fig. 157). Rarely pneumonia
3. In malignant form: inflammation of the respiratory and digestive tract

Judgement : Carcass of an animal showing the clinical disease without secondary complications is conditionally *approved* pending heat treatment. The recovered animals are *approved*. The carcass is *condemned* if the acute febrile or pustular stage of the disease is associated with secondary bacterial infections or if the carcass is inadequately bled. If bacteriological examination showed negative results, this carcass may be *conditionally approved* pending heat treatment.

Differential diagnosis : Contagious ecthyma, scabies, eczema, ulcerative dermatitis and peste des petits ruminants.

Fig. 155: Sheep pox. Lesions on the muzzle and lips.

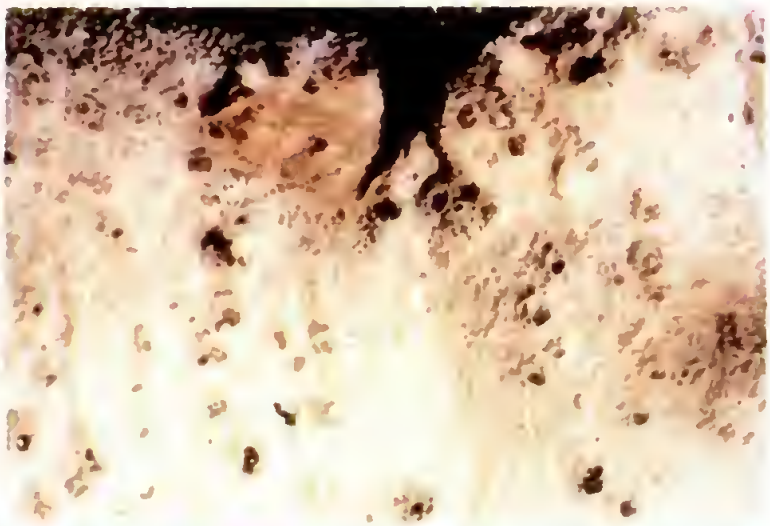


Fig. 156: Sheep pox. Necrosis and coalescing of the lesions and loss of wool.

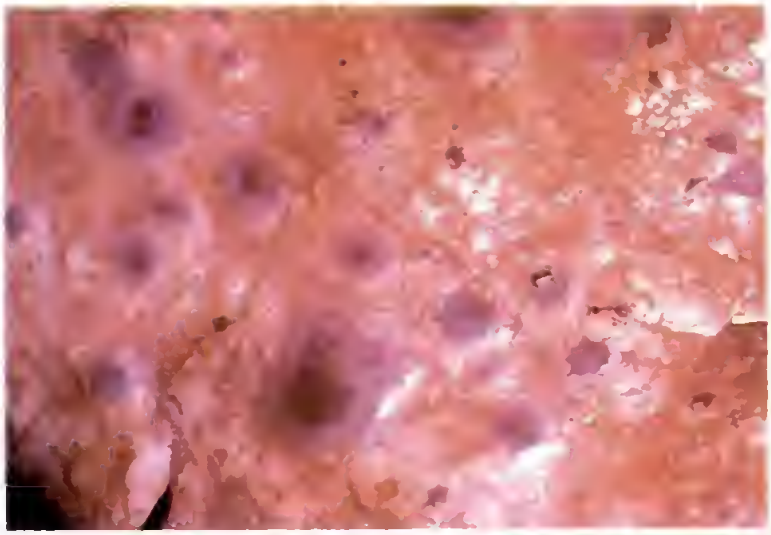


Fig. 157: Sheep pox. Reddish to whitish nodules in the lungs.

Scrapie

Scrapie is a chronic disease of the central nervous system in sheep and occasionally goats characterized by itching, nervous signs and a long incubation period. It is caused by a viral agent called "*viroid*" or "*prion*", which has some of the characteristics of the virus, a "slow" virus like BSE and Maedi.

Transmission : Most likely, the organism enters through breaks in the skin and mucous membranes of susceptible sheep. The agent is present in the lymph nodes, spleen, spinal cord and brain of infected sheep. It is transmitted from sick animals to healthy animals through pasture, where it may be infective for over 3 years. Vertical transmission from the dam and possibly the sire in sheep may also occur. The disease may be transmitted by inoculation of infective material. The agent is resistant to rapid freezing, thawing, boiling for 30 minutes and even to a 20 % formalin solution. At temperatures of 0 - 4°C, the prion is still active after two years. Oscillation of the temperature from 37 - 70°C does not affect its infectivity. At temperatures of 94 - 98°C, the prion is still resistant for 24 hours.

Antemortem findings :

1. Dry wool and rough skin
2. Loss of wool from the head down over the side of the face, rump, thigh, tail base and abdomen
3. Changes of behaviour. Charging of fences, dogs etc.
4. Biting of legs, flanks and belly because of severe itchiness (pruritus)
5. Smacking and rarely curling of the lips and wagging of the tail during rubbing of the skin over the back and sacrum
6. Grinding the teeth
7. Twitching of muscles, excitability and wild expression of the eyes

8. Restless animal, continuously laying down and getting up
9. Incoordinated gait, tendency to run and fall down.
10. Convulsions

Postmortem findings :

1. No gross lesions observed
2. Microscopy reveals the presence of large vacuoles in the cytoplasm of neurons; this is considered a diagnostic lesion.

Judgement : Carcass and viscera affected with the clinical disease are *condemned*. Carcass of contact animals, offspring and ancestors may have a limited distribution or it may be condemned if economically feasible.

Differential diagnosis : Pseudorabies, scabies, thallium poisoning, cobalt deficiency, louping ill, pregnancy toxaemia, external parasitism and photosensitive dermatitis

Fig. 158: Scrapie. Incoordinated gait, twitching of muscles and wild expression in the eyes.

Pulmonary adenomatosis (Jaagsiekte, Driving sickness)

Pulmonary adenomatosis is a chronic progressive pneumonia of sheep with the development of a primary lung neoplasm. This neoplasm is carcinomatous and infrequently metastatic to regional lymph nodes. A *retrovirus* causes the disease and a herpesvirus acts in a secondary role. This is a disease of old ewes, more then 4 years of age. Lambs and yearling are rarely affected.

Transmission : The disease is experimentally transmitted by inhalation of infected droplets by sheep that are kept in close contact. Vertical transmission from pregnant ewes to fetus has also been demonstrated.

Antemortem findings :

1. Incubation 2 months to 2 years
2. Difficult breathing and lacrimation
3. Loss of weight and emaciation
4. When the rear of a sheep is lifted, excess fluid will run from the nose (wheel barrow test).
5. Emaciation and lacrimation

Postmortem findings :

1. The lungs are increased in size and weight (as much as triple their normal size) and do not collapse when the thoracic cavity is opened (Fig. 159).
2. Bluish grey consolidation of the ventral part of the lung
3. Secondary bacterial infections in the lungs
4. Focal lung lesions are interspersed with areas of emphysema.
5. Metastasis of the neoplasm into the bronchial and mediastinal lymph nodes may occur infrequently.

Judgement : Carcass judgement depends on the extent of lung

involvement, condition of the carcass and secondary bacterial infection. Extensive lung lesions with metastasis and loss of musculature *would necessitate the condemnation* of the carcass.

Differential diagnosis : Verminous pneumonia, Maedi/Visna, caseous lymphadenitis and other debilitating diseases

Fig. 159: Pulmonary adenomatosis. Lung lesions showing light grey, enlarged apical and cardiac lobes consisting of numerous greyish coalescing nodules (1 mm to 1 cm in diameter).

Ovine progressive interstitial pneumonia (Maedi, Maedi-visna)

Maedi/visna is a highly fatal viral disease of sheep and goats caused by a *lentivirus*.

Transmission : Through colostrum to newborn lambs and less often by contact with respiratory route.

Antemortem findings :

1. Listlessness
2. Difficult breathing and frequent coughing
3. Nasal discharge
4. Emaciation
5. Lameness
6. In chronic cases, anaemia and secondary bacterial infections

Postmortem findings :

1. Enlarged grey-yellow non collapsible lungs of rubbery consistency (Fig. 160)
2. Cross section of lung parenchyma showing a meaty appearance
3. Enlarged and firm mediastinal lymph nodes

Judgement : Carcass in good flesh with slight to moderate pulmonary involvement is approved. An emaciated carcass with extensive pulmonary lesions or secondary bacterial infection is condemned.

Differential diagnosis : Parasitic pneumonia, pulmonary adenomatosis (Jaagsiekte) and pseudoglanders (Melioidosis)

Fig. 160: Ovine progressive interstitial pneumonia. Cross section of the lung parenchyma. The lungs are enlarged, non collapsible and have a meaty appearance.

Nairobi sheep disease

Nairobi sheep disease is a non contagious, *tick borne viral disease* in sheep manifested by acute haemorrhagic inflammation of the stomach and intestine and by respiratory signs.

Transmission : Adult forms of a tick Rhipicephalus appendiculatus which attach themselves inside the ear of an animal. Unfed adult ticks are infective for one year. Faeces does not contain the virus.

Antemortem findings :

1. Incubation 4 - 15 days
2. Fever; during fever the blood, urine and tissue are infective
3. Rapid painful breathing
4. Dullness and depression
5. Mucopurulent nasal discharge
6. Pain and grunting with defecation
7. Acute haemorrhagic gastroenteritis
8. Bright to dark green faeces (is important in the differential diagnosis.)
9. Abortion in pregnant ewes
10. Swollen vulva and external genitalia
11. Collapse and death

Postmortem findings :

1. Excess fluid in the pericardium
2. Ecchymotic and petechial haemorrhage in the heart muscle
3. Acute haemorrhagic inflammation of the stomachs (Fig. 161) and intestine
4. Distended gall bladder contains thick syrupy bile
5. Enlarged and edematous lymph nodes
6. Hyperaemic genital tract

Judgement : Carcass of an animal affected with the acute disease accompanied with fever and acute gastrointestinal lesions is *condemned*. Carcass of recovered animals and of animals with non systemic or generalized lesions is *approved*. The affected organs are *condemned*.

Differential diagnosis : Rift Valley fever in sheep. Diarrhoea in RVF may show blood tinged watery faeces, but is not green in colour as in NSD. In rinderpest ulcerative lesions are noted with bloody (and not green) faeces. Heartwater, anthrax and plant poisoning should also be considered in differential diagnosis.

Fig. 161: Nairobi sheep disease. Acute haemorrhagic inflammation of the stomachs.

Diseases caused by Mycoplasma spp.

Contagious caprine pleuropneumonia

Contagious caprine pleuropneumonia is a contagious disease of goats caused by *Mycoplasma mycoides subs. capri* (mycoplasma biotype F 38). The disease resembles bovine pleuropneumonia, however it is not transmissible to cattle.

Transmission : By inhalation; carrier or infected animals may also bring the infection into the flock.

Antemortem findings :

1. Incubation: 6 - 10 days
2. Extremely infective with morbidity of 100 %
3. Acute disease with mortality of 60 - 70 %
4. Fever
5. Cough
6. Tongue sticking out and frothy salivation
7. Mouth breathing in terminal stage
8. Lagging and frequently laying
9. Death in few days

Postmortem findings :

1. Fibrinous inflammation of the pleura (Fig. 162)
2. Slight interlobular pulmonary reaction. Lesion may be present in only one lung.
3. Pleural adhesions
4. Enlarged mediastinal lymph nodes
5. No sequestration of necrotic areas as in cattle

Judgement : Carcass of an animal affected with contagious caprine pleuropneumonia which shows no systemic involvement is *approved*. The affected organs are *condemned*. The septicemic form of the disease calls for carcass *condemnation*.

Differential diagnosis : Foot and mouth disease, vesicular stomatitis, shipping fever (pasteurellosis), East Coast fever, foreign body pneumonia, infectious bovine rhinotracheitis, tuberculosis, chlamidial infections and lungworms

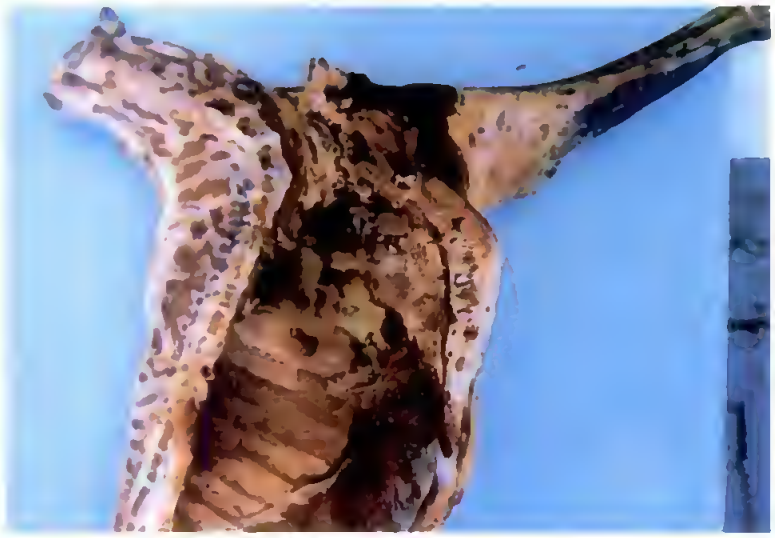


Fig. 162: Contagious caprine pleuropneumonia. Fibrinous inflammation of the pleura.

Diseases caused by bacteria

Brucellosis (see Chapter 3)

Fig. 163A: Brucellosis. Edema and swelling of scrotum.

Black quarter (Black leg)

Black quarter is an acute infectious disease of sheep and cattle manifested by inflammation of the muscles, toxaemia and high mortality. It is caused by Clostridium chauvoei.

Transmission : Contaminated soil. The organisms enter into the digestive tract with feed and through cuts which occur during the shearing, docking, and castration, and via naval infection during birth. Infection of the vulva and vagina of the ewes during lambing may cause serious outbreak of the disease. Black leg is worldwide in distribution. Well nourished and grass fed animals are more often affected.

Antemortem findings :

1. Fever
2. Loss of appetite
3. Depression
4. Stiff gait and reluctance to move due to lameness
5. Subcutaneous edema is not common.
6. Gaseous crepitation occurs before death.
7. Head lesions associated with edema and nose bleeding

Postmortem findings :

1. Subcutaneous edema particularly noted around head.
2. Affected muscle is dark brown, dry and sponge like or moist. A pungent odour is noted. Less gas is formed than in cattle.
3. Tongue, heart muscle and/or diaphragm may be blackish red. Marked abdominal extension if fetus is infected.
4. Genital tract lesions in the walls of the vagina and occasionally uterus
5. Serosanguineous and haemorrhagic fluid in body cavities and

pericardial sac
6. Edema of lungs

Judgement : *Total condemnation* of the carcass and viscera of an animal affected with black leg. It is prohibited to slaughter and dress the animal diagnosed with this disease on antemortem examination.

Differential diagnosis : Other acute Clostridial infections, lightning strike, anthrax, bacillary haemoglobinuria, malignant edema, extensive haemorrhage, acute lead poisoning and lactation tetany

Enterotoxaemia (Pulpy kidney)

This disease is a fatal toxaemia in lambs, sheep, goats, calves and seldom in adult cattle. The disease is manifested by diarrhoea, involuntary contraction of muscles, paralysis and sudden death. It occurs after a sudden change to a better, more nutritious diet. The disease is often noted in sheep that have been fed heavy grain, and in animals which graze on lush growing pastures. *Clostridium perfringens* multiplies in abomasum and intestine and produces toxin which paralyses the vital centres in brain and damages endothelium of blood vessels. The disease occurs extensively in particular in Southern Africa but is well controlled by vaccination.

Antemortem findings :

1. Short course of the illness (2 - 12 hours) in lambs and longer course (24 hours) in sheep
2. Animal found dead without previous sign of the disease
3. Dullness and depression
4. Rapid shallow respiration

5. Loss of appetite and frothing
6. Muscular contractions
7. Green pasty diarrhoea
8. Grinding of the teeth and muscular tremor
9. Logging behind the flock
10. Staggering and recumbency

Postmortem findings :

1. No lesions in peracute cases
2. Large amount of clear, straw coloured pericardial fluid
3. Petechial haemorrhages of the heart muscle
4. Congestion of the abomasal and intestinal mucosa (Fig. 163) and liver
5. Soft pulpy kidneys a few hours after death is characteristic of this disease
6. Overload of the rumen and abomasum with concentrate
7. Haemorrhage and edema in sheep brain
8. Rapid decomposition of the carcass

Judgement : Carcass of an animal affected with enterotoxaemia is *condemned*.

Differential diagnosis : Sudden death in lambs: pasteurellosis, hypocalcemia and hypomagnesemia (reduced blood calcium and magnesium), polioencephalomalatia (less acute form), acute rumen impaction (no convulsions are present and the course is longer) and other septicemias. Adult sheep: rabies, acute lead poisoning, pregnancy toxaemia and louping-ill

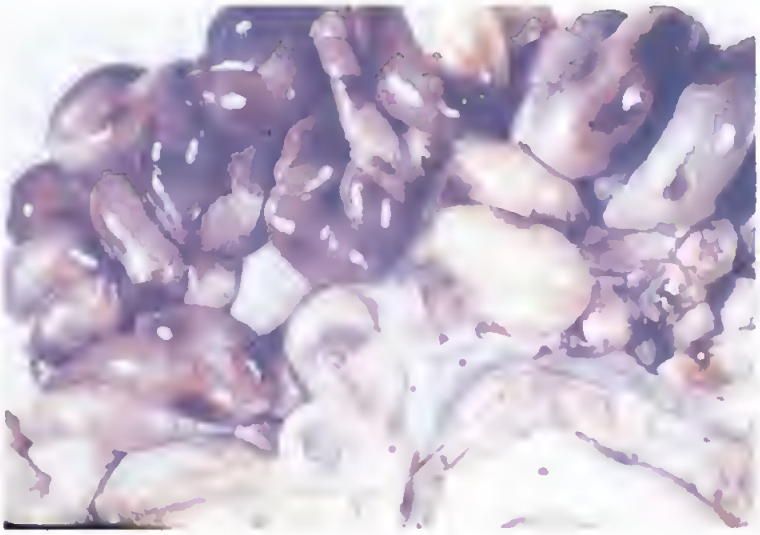


Fig. 163: Enterotoxaemia (pulpy kidney). Dilated intestine showing a patchy congestion. Note also congestion of mesenteric lymph nodes.

Infectious necrotic hepatitis (Black disease)

Black disease causes acute necrotic hepatitis in sheep and cattle and rarely in pigs. It is caused by bacterium *Clostridium novyi* in association with immature fluke invasion of the liver.

Antemortem findings :

1. Fever (40 - 42°C)
2. Rapid and shallow respiration
3. Sheep may be found dead without clinical signs.
4. Sick animal usually segregates from the rest of the flock.
5. Depression and incoordination
6. Recumbency

Postmortem findings :

1. Dark brown swollen liver showing necrotic areas surrounded by a zone of hyperaemia (Fig. 164)
2. Evidence of recent infestation of liver flukes

3. Darkened and cyanotic subcutaneous tissue due to small blood vessel engorgement (dark appearance of the skin). The name "Black disease" was derived from this.
4. Clear straw coloured fluid in the abdominal and thoracic cavities and in the pericardial sac

Clostridium novyi is an endemic environmental contaminant and remains latent in the liver, spleen and bone marrow. Immature liver flukes, by migrating through the liver, cause liver necrosis. This initiates Cl. novyi spores to germinate and proliferate. Necrotizing and haemolytic toxins are produced which cause generalized toxaemia and haemolysis of the blood.

Judgement : Carcass and viscera affected with black disease are *condemned*.

Differential diagnosis : Fascioliasis, enterotoxaemia, blackleg, malignant edema and anthrax

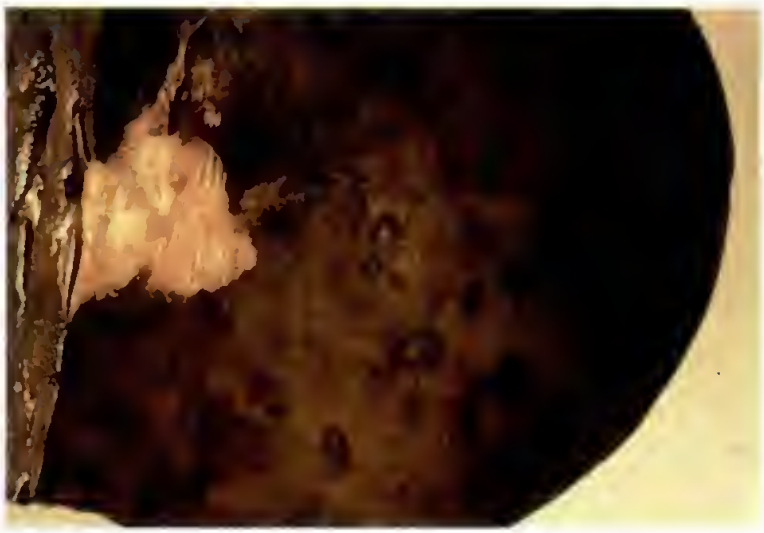


Fig. 164: Black disease. Dark brown swollen liver showing necrotic areas (1-2 cm) in diameter surrounded by a zone of hyperaemia.

Caseous lymphadenitis

This is a chronic disease of sheep and goats manifested by abscesses in the lymph nodes. It is caused by *Corynebacterium pseudotuberculosis*. Caseous lymphadenitis has a worldwide distribution and causes great economic losses to the sheep industry.

Transmission : Discharge from the lymph nodes, via wounds caused by shearing, castration and docking, contaminated sheep dips, skin abrasions or traumatized oral mucosa. Animals with open abscesses should be segregated in order to prevent the spread of the disease.

Antemortem findings :

1. Animal is lagging behind the flock.
2. Dyspnea
3. Purulent ocular and nasal discharge
4. Enlarged superficial body lymph nodes
5. Generalized disease is associated with weight loss, depression and loss of appetite.

Postmortem findings :

1. Caseous abscesses in the superficial lymph nodes and carcass musculature (Fig. 165)
2. Firm and dry abscess in the kidney (Fig. 166) and other organs Soft pasty abscess in the early stages changes to firm and dry with a characteristic laminated appearance in the later stages of disease.
3. Abscess content is creamy and pasty in goats
4. Pneumonia

Differential diagnosis : Abscesses in the organs and viscera,

neoplasm, echinococcosis and other parasitic lesions

Judgement : If this condition is associated with extensive involvement of many lymph nodes and tissues, suggesting a haematogenous spread, the carcass is *condemned*. Otherwise it is *approved*. The affected tissue is *condemned*.

Remarks : An abscess in a body lymph node is a sequel to the organism gaining entrance into the body via skin wounds etc. The drained area of the lymph node should be examined. If no other lesions are observed, it may be an indication that the lymph node has sequestered the agent. It is not necessary to condemn a quarter or a carcass due to a lesion in one lymph node or in several lymph nodes.

Fig. 165: Caseous lymphadenitis. Caseous abscess filled with greenish-yellow pus in the abdominal muscles.

Fig. 166: Caseous lymphadenitis. Firm and dry abscess in the kidney.

Parasitic diseases

Diseases caused by helminths

Coenurus cerebralis infection (Gid, Sturdy)

Coenurosis is a disease of the brain and spinal cord caused by the intermediate stage of *Taenia multiceps* which inhabits the intestine of dogs, cats and wild carnivores. The clinical disease occurs in sheep and rarely in cattle.

Life cycle : *Eggs* expelled with dog faeces are ingested by the intermediate host (sheep). The *larvae* hatch in the intestine and pass with the blood stream towards different organs. The larvae which reach the brain and spinal cord grow to the *coenurid* stage. Coenurus cerebralis will further mature in the brain and spinal cord.

Antemortem findings:

During migration of larval stage

1. Blindness
2. Muscular tremor and incoordination
3. Excitability and collapse

Infection with the fully developed larval stage

4. Salivation
5. Wild expressions
6. Frenzied running and convulsion
7. Deviation of eye and head
8. Loss of function
9. Dullness
10. Incomplete mastication
11. Head pressing
12. Incomplete paralysis and, in spinal cord involvement, inability to rise

Postmortem findings :

1. Thin walled cyst in the brain (Fig. 167)
2. Lesion in the lumbar region and rarely, in the cervical area of the spine

Fig. 167: Coenurus cerebralis. Thin walled cyst in the brain.

Judgement : Carcass affected with coenurosis is *approved.* Affected brain and organs are *condemned.*

Differential diagnosis : Abscess, haemorrhage, brain tumours and in early stages, inflammation of the brain and rabies

Echinococcosis (Hydatid disease)

Hydatid disease occurs in sheep, cattle, swine, horses and humans. Echinococcosis is a disease which occurs when the larval stage of *Echinococcus granulosus* and *Echinococcus multilocularis* are ingested by an intermediate host (sheep, cattle). These larvae then develop into hydatid cysts in various tissues. The adult tape worms are found in dogs, cats and other carnivores. They may ingest the hydatid cysts by eating infected organs of the intermediate hosts. The scolex attaches to the intestinal wall. Adult tapeworms develop in approximately seven weeks and *eggs* are shed in the faeces and are ingested by sheep and cattle. The ova hatch to liberate the *onchospheres* which penetrate the intestinal wall and through the portal venous supply to the liver where they become arrested. In older sheep and cattle the larvae may reach the lungs and various other organs through the systemic circulation. The most common sites of cysts are the liver and lungs. The *cysts* are different sizes and shapes and they contain a clear fluid. Due to the growth of the cyst, pressure atrophy is noted in the surrounding tissue.

Daughter cysts are found outside the mother cyst and are formed due to trauma or external pressure on the mother cyst. They may or may not be attached to the mother cyst. Daughter-cyst formation may have neoplastic characteristics when there is penetration to the blood and lymph vessels and metastases to various distant organs.

Humans gets infected with hydatid disease via the ingestion of ova from Echinococcus tapeworm in the dog. This usually occurs by touching dog hair that has been contaminated by ova from faeces. It also may occur by the dog transferring ova from the anus to its mouth and then by licking humans.

Postmortem findings : Multiple Echinococcus granulosus cysts in the liver, lungs (Fig. 168) and other organs.

Judgement : The animal carcass affected with echinococcosis is *approved* if edema and emaciation are not found. Otherwise the carcass is *condemned*. The affected organs are also *condemned* and must be destroyed. The lungs are most commonly affected and these should be carefully checked because lesions are often missed on routine inspection.

Differential diagnosis : C. tenuicollis, C. cellulosae, calcified TB lesions and congenital cysts

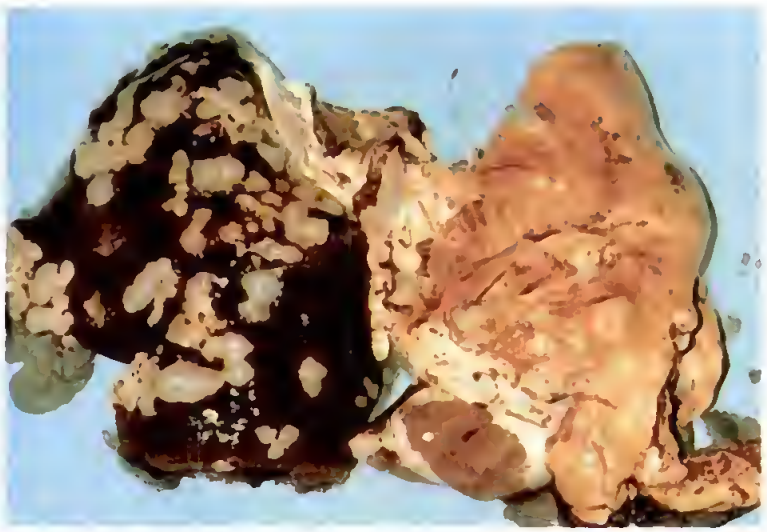


Fig. 168 : Echinococcosis. E. granulosus cysts in the liver.

Lung worms

Dictyocaulus filaria is the common sheep lung worm which cause verminous pneumonia or bronchitis.

Life cycle: (Fig. 169) Adult worms live in the bronchi where they lay *eggs* which are coughed up to pharynx and swallowed by the host. The eggs are hatched in the digestive tract and the *larvae* are then expelled in the faeces. In a moist environment and moderate temperature, the larvae will become infective in 3 - 7 days. Larvae are resistant to cold, although it will cause their maturation to be delayed. Upon digestion by sheep (primary host), larvae penetrate the intestinal wall and reach the mesenteric lymph nodes. From the mesenteric lymph nodes via the blood stream, larvae migrate to the lung alveoli and further to the bronchi. They *mature in the bronchi* and lay eggs. The cycle is then repeated.

Muellerius capillaris parasitises in the alveoli and pulmonary parenchyma. Intermediate hosts are snails and slugs which sheep ingest during grazing. Larvae reach the lungs and produce small greyish nodules on the back of the lungs.

Antemortem findings :

1. Difficult breathing
2. Cough and nasal discharge
3. Fever if secondary infection present

Post mortem findings :

1. Exudate in bronchioles and resulting collapse of lung portion
2. Verminous pneumonia with consolidation of lung parenchyma
3. Enlarged lung lymph nodes
4. Greyish-green nodules encysted or calcified with Muellerius

capillaris infestation (Fig. 170)

Judgement : Carcass is *approved* in lung worm infestation if no secondary changes are observed. The lungs are *condemned*. If lung worm infestation has caused pneumonia, emaciation or anaemia, the carcass is *condemned*.

Differential diagnosis : Bacterial bronchopneumonia, abscess, necrobacillosis, tuberculosis, actinobacillosis, hydatid disease and atelectasis

Fig. 169: Life history of lungworm of sheep

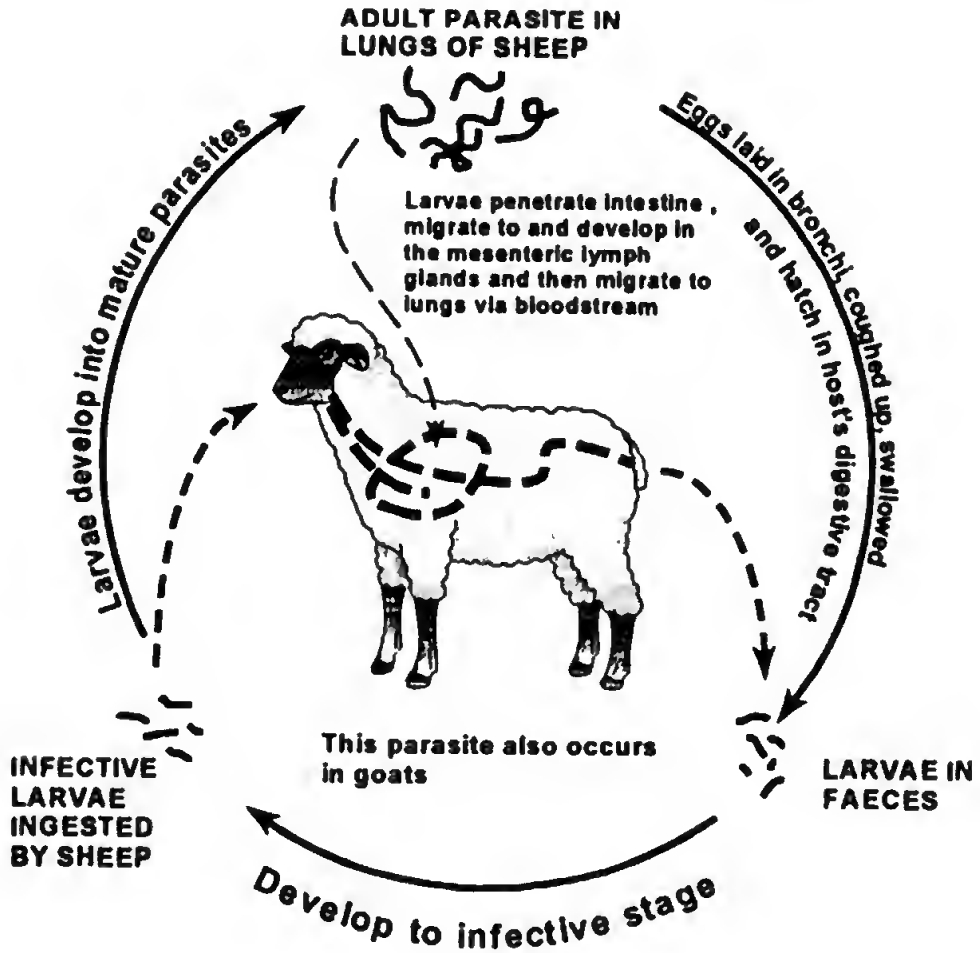


Fig. 170: Lung worms. Numerous greyish nodules of M. capillaris in the lung parenchyma.

Fascioliasis

The fluke *Fasciola hepatica* is most frequently found in sheep and cattle and less often in goats and swine. Acute fascioliasis occurs almost entirely in sheep. In sheep and cattle, wandering flukes damage liver tissue and bile ducts which then become thickened and fibrous.

Life cycle : The adult flukes of Fasciola hepatica are found in the bile ducts and gall bladder. The eggs are shed into the bile duct from which they pass to the intestine. With animal faeces, the eggs are expelled out on the pasture.

The larvae (*miracidia*) enter aquatic snails (Limnea truncatula) which are the intermediate hosts and develop into *sporocysts* and later into *rediae*. The rediae will further develop into the final larval stage (*cercaria*). Cercaria will transform in the external environment to *metacercaria*. If ingested by herbivorous animals, metacercaria will penetrate the small intestinal wall, cross the

peritoneal space and reach the liver. In the bile ducts, metacercaria will mature into an *adult fluke*. The metacercariae which do not reach the bile ducts will encapsulate in the liver parenchyma.

Fasciloides magna is a large liver fluke which is prevalent in elk, deer and moose. Sheep and goats are susceptible to infection if they share the pasture with those wild animals. F. magna in sheep continuously migrate through the liver parenchyma and may cause death in less than six months.

Lancet flukes (*Dicrocoelium dendriticum*) in sheep cause little damage to the liver parenchyma except for a moderate to marked thickening of the bile ducts.

Antemortem findings :

1. Weight loss
2. Anaemia and edema
3. Chronic diarrhoea

Postmortem findings :

1. Black parasitic debris in the liver (Fig. 171), lungs, diaphragm and peritoneum
2. Black lymph nodes of the lungs and liver due to fluke excrement

Judgement : Carcass of an animal affected with fascioliasis is *approved* if in good flesh and emaciation and edema are not observed. A heavily infested parasitic liver is *condemned*.

Differential diagnosis : Nutritional deficiencies of copper and cobalt, infectious necrotic hepatitis, black disease, anthrax, enterotoxaemia, melanosis, melanoma

Fig. 171: Fascioliasis. Black parasitic debris in the liver.

Cysticercus tenuicollis infestation

Cysticercus tenuicollis is the cystic stage of tape worm *Taenia hydatigena* which is found in dogs and cats. *Ova* pass with dog faeces on the pasture and may get ingested by intermediate hosts sheep and pigs. *Larvae* which develop from ova penetrate the intestine and pass by portal vein to various tissues especially the omentum, mesentery, peritoneum and liver. Migration through the liver leaves greyish-white tortuous tracts. If larvae reach the liver surface they develop into thin-walled fluid filled bladders and if they fail they degenerate and become calcified.

Heavy infestation with Cysticercus tenuicollis in young animals causing liver damage and haemorrhages or peritonitis, rarely results in the death of the animal.

Antemortem findings :

Moderate to heavy infections produce:-

1. Loss of appetite
2. Depression
3. Weakness

Postmortem findings :

1. Cysts of different diameters on the liver, diaphragm and peritoneum
2. Subserosal cysts on the liver (Fig. 172)

Judgement : The carcass affected with cysticercus tenuicollis is *approved*. The organs are *condemned* and affected serous membranes should be stripped.

Differential diagnosis : C. bovis, C. cellulosae, hydatid cysts and calcified TB lesions

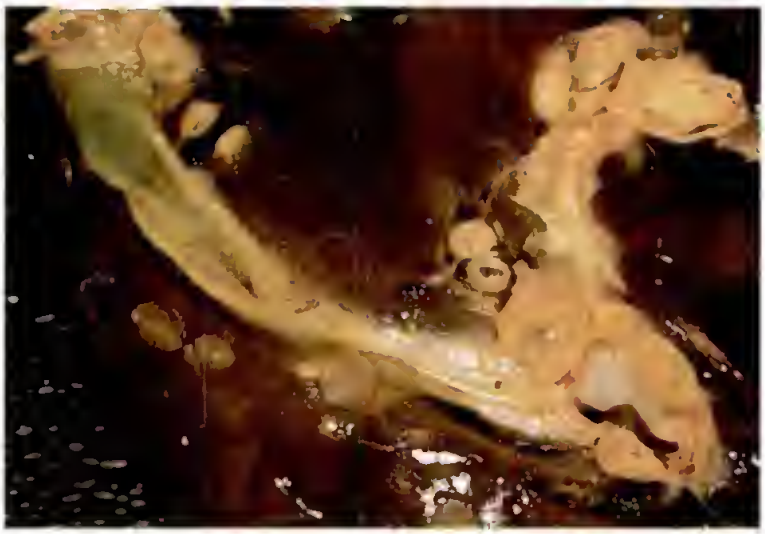


Fig. 172: Cysticercus tenuicollis. Numerous subserosal cysts in the liver.

Cysticercus ovis infestation (sheep measles, sheep bladder worm)

Cysticercus ovis is the larval stage of *Taenia ovis*, a tapeworm found in the intestines of dogs and wild carnivores. Its development is similar to that of *Taenia saginata*. However, in the case of *Taenia ovis*, the definitive hosts are *sheep*. The cysts are found in the heart, diaphragm, masseters and the skeletal musculature of sheep. They are fully developed from 7 to 10 weeks after the ingestion of the ova. The rapid degeneration of cysts commence almost immediately after the cysts reach maximum development. When degenerated, the cysts appears as a caseous nodule in the musculature.

Antemortem findings : Usually no clinical signs are recognized.

Postmortem findings :

1. The cysts are oval, measure 9 mm × 5 mm when fully developed and are most common in the heart (Fig. 173), the masseters, the diaphragm and the skeletal musculature (Fig. 174).
2. In older animals the cysts degenerate and calcify
3. The degenerated cysts appears as greenish yellow caseous nodules with calcification often present.

Judgement : In moderate or light infestation consisting of a small number of dead or degenerated cysticerci, the carcass can be *boned out* under supervision, the *cysts removed* and the meat passed after being held for 10 days at -10°C. If the *freezing treatment* is not possible, the *heating* of the carcass at 56°C is suggested.

In heavy infestations the carcass is *condemned*. It is commonly considered that an animal is heavily infested if lesions are

discovered in two of the usual inspection sites including the masseter muscle, tongue, oesophagus, heart, diaphragm or exposed musculature and in two sites during incision into the shoulder and the rounds. Carcasses with C. ovis infestations may not be acceptable for export.

Differential diagnosis : Sarcocystosis, eosinophilic myositis, neurofibromatosis, abscesses, C. tenuicollis, caseous lymphadenitis

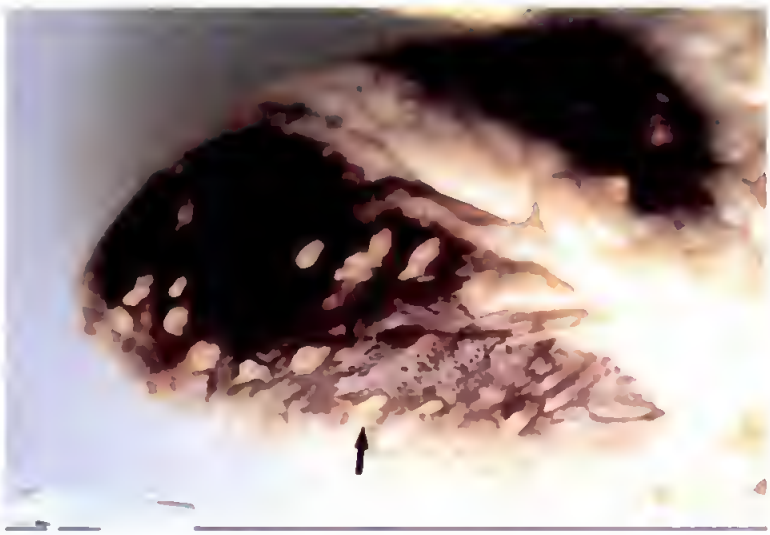


Fig. 173: Cysticercus ovis. The heart of an old ewe showing heavy infestation with C. ovis. The cysts have degenerated and undergone calcification. (Courtesy Dr. D. Baucks)

Fig. 174: Skeletal muscles of a sheep with several cysts of C. ovis undergoing calcification. (Courtesy Dr. D. Baucks)

Stilesia hepatica

This is a tape worm which occurs in the bile duct of sheep, goats and wild ruminants. The life cycle is not completely known but oribatid mites are suspected of transmitting the parasites. The parasite affects animals of all ages and is considered non pathogenic. Heavy infections are frequently seen in apparently healthy sheep. With almost complete occlusion of the bile ducts, icterus and the other clinical signs are not observed. There are areas where approximately 80 % of sheep and goat livers are affected.

Judgement : The carcass is *approved* unless associated with emaciation. The affected liver is *condemned*. In some parts of the world, all sheep livers are condemned on postmortem inspection, because of high rate of liver infections.

Fig. 175: Stilesia hepatica. Long, threadlike parasite (20-50 cm long and up to 3 mm wide) in the sheep liver.

Diseases caused by protozoa

Babesiosis (Piroplasmosis, Texas fever, Red water, Tick fever)

Babesiosis is a protozoan parasitic febrile disease of cattle, horses, sheep and swine caused by Babesia spp..

In sheep and goats, babesiosis is caused by *Babesia motasi* and *Babesia ovis*. Acute signs of the disease are characterized with fever, anaemia, parasitemia and haemoglobinuria. B. ovis usually causes a milder form of the disease than does B. motasi. The parasite grows and multiplies in the blood corpuscles (erythrocytes) of sheep and goats and causes haemoglobin (constituent of erythrocytes) elimination in urine (haemoglobinuria).

Transmission : Different species of ticks in the family Ixodidae serve as vectors of infection. Babesia ovis infection transmitted experimentally in sheep has caused acute signs of disease, parasitemia and lasting immunity similar with babesiosis in cattle.

Antemortem findings :

1. Incubation 7 - 10 days
2. High fever (41.5°C)
3. Difficult breathing
4. Anaemia
5. Loss of appetite
6. Dark reddish brown urine
7. Recovered animals may be emaciated, have reduced milk production, and some may also abort.

There are no characteristic signs in the chronic disease.

Postmortem findings :

1. Enlarged, yellow liver and distended gall bladder containing thick dark bile. The bladder mucosa is edematous and yellow.
2. Subcutaneous tissue and connective tissue in the muscles are edematous and jaundiced.
3. Thin watery blood and red urine in the bladder
4. Enlarged spleen
5. Edematous and haemorrhagic lymph nodes

Judgement : Carcass of an animal in the subclinical form of the disease or in the chronic stage may have a *favourable judgement* providing the carcass is adequately set and icterus is not present. An animal carcass showing acute form of the disease accompanied with fever, marked anaemia and haemoglobinuria and/or emaciation is *condemned*.

Differential diagnosis : Trypanosomiasis, theileriosis, haemobartenellosis, leptospirosis, bacillary haemoglobinuria and anaplasmosis

Toxoplasmosis

Toxoplasmosis is a contagious disease of animals and man caused by protozoon *Toxoplasma gondii*. It is found most frequently in pigs and sheep. Toxoplasma in sheep is manifested with abortion and stillbirths in ewes.

Life cycle : see Fig. 147

Antemortem findings :

1. Abortion and stillbirths in ewes
2. Fever
3. Generalized tremor
4. Difficult breathing

The systemic disease is seldom found in sheep.

Postmortem findings :

1. Multiple granulomatous lesion in the lungs
2. Hydrothorax
3. Ascites
4. Intestinal ulceration
5. Necrosis in the liver, spleen and kidneys
6. Necrosis of placenta
7. Brain haemorrhage, edema and ventricular dilatation (Fig. 176)
8. Inflammation of the brain (Fig. 177)

Judgement : Carcass of an animal showing clinical signs of acute disease is *condemned*. Recovered and reactor animals are *approved*.

Differential diagnosis :

Abortion in ewes: brucellosis, campylobacteriosis, listeriosis, salmonellosis and Rift Valley fever

Brain lesions: salt poisoning, chlorinated hydrocarbons, lead, mercury, Vitamin A deficiency, hypoglycaemia, encephalomalacia, meningitis, rabies and scrapie

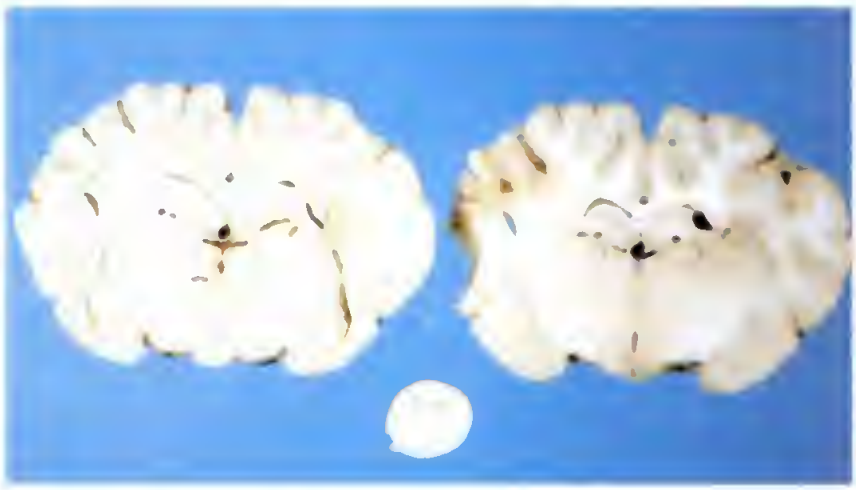


Fig. 176: Toxoplasmosis. Brain haemorrhage, edema and ventricular dilatation. The specimen was fixed in 10% formalin solution.

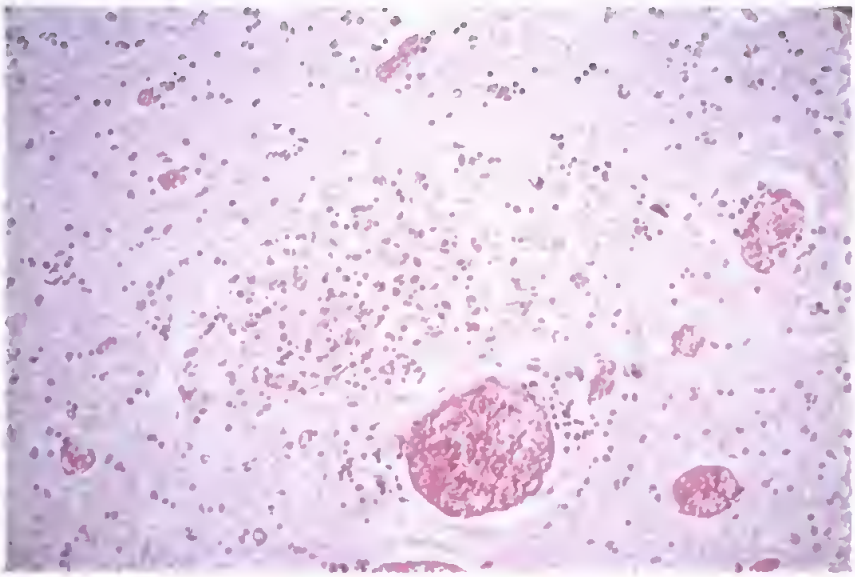


Fig. 177: Toxoplasmosis. Inflammation of the brain (encephalitis). Tachyzoites are distributed throughout the brain where they encysts and produce bradyzoites.

Theileriosis (Malignant ovine or caprine)

Theileriosis is thick borne disease of sheep and goats, cattle, buffalo and wild ruminants caused by species of protozoa in the genus Theileria. In sheep and goats, the infections are caused by T. hirci and T. ovis. *Theileria hirci* is the cause of an acute and highly fatal disease of sheep and goats in Eastern Europe, the Middle East, Asia and North Africa. The subacute and chronic forms have also been reported. Mild infection in noted young lambs and kids. *Theileria ovis* causes a milk disease in sheep and goats; a disease from which they rapidly recover.

Transmission : The thick vector is unknown in *Theileria hirci* infection, although Hyalomma spp. are suspected.

Antemortem findings :

In acute form

1. Morbidity rate of 100 % and mortality of 46 - 100 %
2. Fever (40°C - 41°C)
3. Loss of appetite and listlessness
4. Increased heart rate and difficult breathing
5. Edema of the throat and subsequent death
6. Hyperaemia of the conjunctiva and nasal discharge
7. Swollen superficial lymph nodes
8. Atony of the rumen in the chronic form
9. Mild fever, anaemia, icterus, weakness and emaciation

Postmortem findings :

1. The lesions are basically similar as those observed in bovine Theileriosis (T. parva).
2. Edema of the lungs
3. The yellowish-brown liver may be increased in size and shows soft and friable consistency.

4. Enlarged haemorrhagic lymph nodes and enlarged spleen
5. Kidney infarcts
6. Petechial haemorrhage in subcutaneous, subserosal and submucosal tissue

Judgement : Carcass and viscera of an animal showing clinical signs of chronic theileriosis and being without gross lesions, are *approved*. If the acute form of the disease is accompanied with fever, icterus and generalized lesions, the carcass and organs are *condemned*.

Differential diagnosis : Babesiosis, Rift Valley Fever and catarrhal fever of sheep

Sarcocystosis in sheep (Sarcosporidiosis)

Sarcocystosis of sheep is a widespread infestation caused by four species of *Sarcocystis* (Table 2). Nearly all adult sheep in most parts of the world are infested. Three other species of Sarcocystis have been described from goats. Their prevalence and importance in meat inspection are not fully known.

The general pattern of the life-cycle is similar to that described for Sarcocystis cruzi in cattle except that each species uses its own definitive hosts. S. tenella and S. gigantea cause the most widespread infestations. S tenella produces microcysts and are the most pathogenic. S. gigantea produces macrocysts and are generally not pathogenic but because of their large size they are important in meat inspection.

Table 2: Sarcocystis spp. in sheep

Species	Distribution	Definitive Hosts	Size and Shape of Cyst	Pathogenicity
Sarcocystis tenella	World-wide	Dog, coyote and red fox	Microscopic, up to 0.7 mm long, may be found in the central nervous system	Pathogenic. Causes anorexia, weight loss, anaemia, fever, abortion and even death. It is the most pathogenic sheep Sarcocystis sp.
Sarcocystis gigantea	World-wide	The domestic cat	Macroscopic, oval or elongated and measures up to 1cm long, More common in order sheep.	Only mildly pathogenic.
Sarcocystis arieticanis	Europe, Australia, New Zealand and the USA	Dog	Microscopic, up to 0.9mm long.	They are less pathogenic than S. tenella
Sarcocystis medusiformis	Australia and New Zealand	Cat	Macroscopic, filiform and elongated up to 8mm long and 0.2mm wide.	Pathogenicity not known.

Antemortem findings (in S. tenella infection):

1. Fever
2. Anaemia
3. Loss of appetite and weight loss

4. Retarded growth
5. Enlarged lymph nodes
6. Abortion
7. Nervous signs

Postmortem findings (in S. gigantea infestations) :

1. Oval, elongated or fusiform cysts up to 1 cm long and 0.5 cm wide in the oesophagus (Fig. 178), pharynx, diaphragm, skeletal musculature, tongue and heart
2. In S. tenella infestations haemorrhages in the serous surface of the viscera, cardiac and skeletal muscles
3. Serous atrophy of pericardial and perirenal fat

Judgement : In heavy infestations the carcass is *condemned*. In moderate to light infestations the lesions are removed and the carcass is *passed*.

Differential diagnosis : Myositis, cysticercosis, grass seeds, necrotic lesions

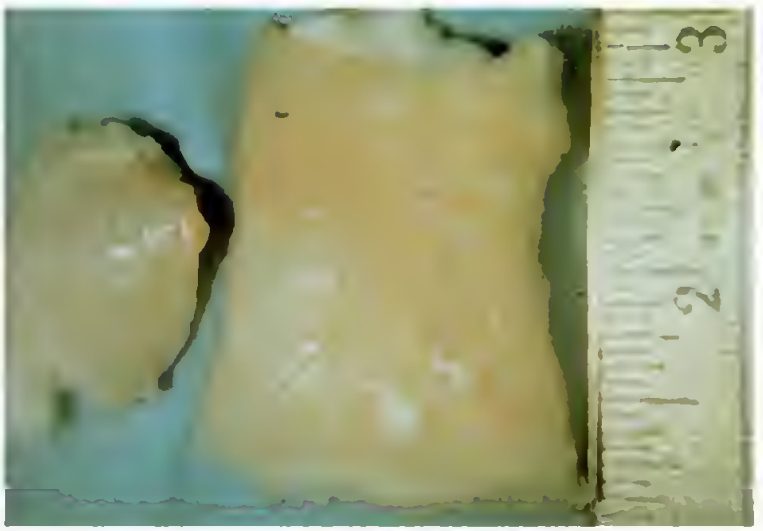


Fig. 178: Sarcocystis gigantea in the oesophagus of a sheep. They resemble cooked rice grains.

CHAPTER 6

SPECIFIC DISEASES OF HORSES

Diseases caused by viruses

African horse sickness (AHS)

AHS is a highly fatal insect-borne febrile viral disease of equidae characterized by edema of the subcutaneous tissue and lungs, haemorrhage and serous fluid in the body cavities. At room temperature, the virus is persistent for a few months in urine, dried blood, faeces and serum. It is resistant to boiling up to 15 minutes, and to common disinfectants. It is destroyed by sun light.

Transmission : AHS is transmitted by various Culicoides spp. and several species of mosquitoes. Mechanical transmission by biting flies is also possible. Dogs get infected by eating infected horse meat.

Antemortem findings :

1. Incubation 3 - 5 days

Cardiac form

2. Mortality up to 50 %
3. Fever up to 40°C
4. Conjunctivitis
5. Non pitting-edema in the supraorbital fossa (above the eye, Fig. 179) is the most characteristic sign.
6. Subcutaneous edema of the head, neck, brisket, thorax and ventral abdomen

Pulmonary form

7. Sudden onset and increased temperature (41°C)
8. Respiratory distress and associated coughing
9. Frothy exudate from the nares for several hours prior to death
10. Mortality is over 90 %

In some animals both forms may occur.

Mild form "horse sickness fever"

11. Fever in duration of a few days
12. Slightly laboured breathing
13. Loss of appetite

Postmortem findings :

1. Petechial haemorrhage on the ventral surface of the tongue
2. Gelatinous edema in the periorbital tissue, neck muscles and ligamentum nuchae (Fig. 180)
3. Intermuscular and subcutaneous edema and haemorrhage
4. Trachea and bronchi filled with froth
5. Pleural exudate and pulmonary edema (Fig. 181)
6. Inflammation of the heart and haemorrhage on the pericardium
7. Excessive pericardial fluid
8. Congestion of the fundic portion of the stomach
9. Haemorrhage of the intestinal serosa
10. Haemorrhagic kidneys

Judgement : Carcass of an animal affected with acute AHF showing generalized clinical signs and postmortem lesions is *condemned*. The carcass of recovered and reactor animals is *approved* for limited distribution.

Differential diagnosis : Colics, anthrax, equine rhinopneumonitis,

equine infectious anaemia, equine viral arteritis, equine piroplasmosis, equine influenza

Fig. 179: AHS. This horse is near death. The animal is depressed and showing edema of supraorbital fossa, neck and chest.

Fig. 180: AHS. Subcutaneous and intermuscular edema in the neck. This may be the only lesion in some cases of AHS.

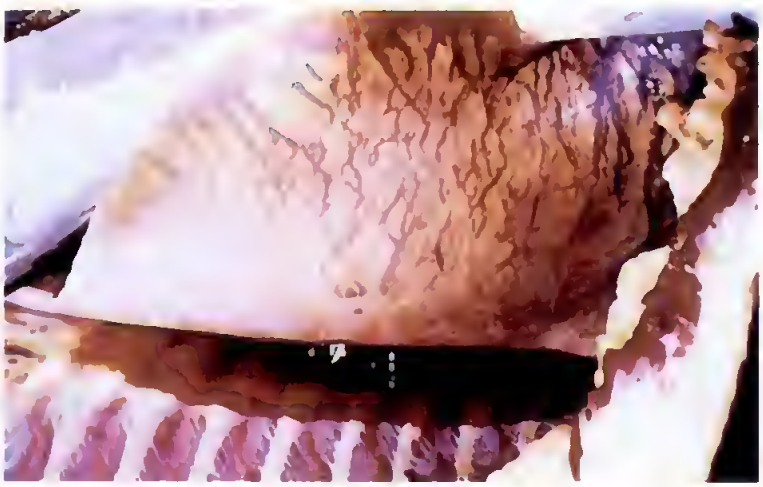


Fig. 181: AHS. Thoracic lesion. Pleural exudate and pulmonary edema are noted.

Equine infectious anaemia

Equine infectious anaemia is an acute, subacute, chronic and latent disease of solipeds caused by a virus. It is manifested with intermittent fever, depression, weakness, edema, anaemia and icterus.

Transmission : Close continued contact with susceptible animals and biting insects mainly Tabanidae and mosquitoes. Contaminated surgical instruments, needles, contaminated feed, bedding and intrauterine infection. Horses may die of anaemia during early viraemia or may recover and have recurrent episodes of viraemia.

Antemortem findings :

Acute form

1. Fever up to 41°C; it may rise and fall rapidly
2. Sudden onset of disease lasting from 3 days to 3 weeks
3. Depression and weakness
4. Jaundice
5. Edema of the ventral abdomen, legs and prepuce
6. Serosanguineous nasal discharge
7. Abortion in pregnant mares
8. Rapid dehydration
9. Droopy ears and half closed eyes
10. Dyspnea at terminal stages

Subacute form

11. An acute onset of disease and subsequent recovery
12. Swollen icteric conjunctivae
13. Exertion may cause an increase in temperature and pulse, sweating and incoordination.
14. Death caused by exhaustion

Chronic form

15. Anaemia, weakness
16. Icteric conjunctivae and mucous membranes
17. Diarrhoea
18. Recurrence of disease in a one to three month period

Latent form

19. No symptoms observed although the animal is infected
20. May become active. An acute attack may be caused by hard work, poor diet and parasitic infections.

Postmortem findings :

Acute case

1. Subcutaneous edema
2. Jaundice
3. Subserosal haemorrhage

Chronic case

4. Emaciation and anaemia
5. Hydrothorax and ascites
6. Enlarged spleen and liver (Fig. 182) with swelling of the edges
7. Superficial haemorrhages in the organs
8. Enlarged, edematous and haemorrhagic lymph nodes
9. Replacement of bone marrow fat with dark red hemopoietic tissue (Fig. 183)

Judgement : Carcass of an animal affected with the clinical disease of EIA is *condemned*. Reactor animals may have the carcass approved for limited distribution if no systemic lesions are noted on postmortem examination.

Differential diagnosis : Emaciation, other acute septicemias, anthrax, piroplasmosis, glanders, tuberculosis, virus encephalomyelitis, purpura haemorrhagica, babesiosis,

leptospirosis, parasitic infections (strongylosis and fascioliasis) and purulent infections causing anaemia

Fig. 182: Equine infectious anaemia. Enlarged grey red liver showing lobular pattern and haemorrhage under the capsule.

Fig. 183: Equine infectious anaemia. Replacement of bone marrow fat with dark red hemopoietic tissue (erythroid hyperplasia).

Viral encephalomyelitis of horses

Viral encephalomyelitis of horses is characterized by disturbed consciousness, motor irritation and commonly high mortality. The disease has been found in cattle and sheep as well as in humans.

Transmission : The disease is mostly spread from birds through insects to horses and humans. Mosquitoes of the Culex, Aedes and Mansonia genera are vectors of this disease. Wild birds are reservoirs of infection.

Antemortem findings :

1. Incubation 1 - 3 weeks
2. Fever
3. Depression and anorexia

Nervous signs

4. Hypersensitivity to sounds and touch, fascial muscle twitch and walking blindly into objects or in circles
5. Paralysis, collapse and death between 2 - 4 days of the onset of symptoms
6. Mouth and eyes may be closed.

Postmortem findings :

1. Gross lesions are not usually found.
2. Visceral haemorrhages
3. Lesions in the lungs and rarely other organs
4. Histological findings include the lesions in the brain showing perivascular accumulation of leucocytes and damage to neurons

Judgement : Carcass and viscera of the animal showing clinical signs of this disease are *condemned*. The carcass of reactor animal may have a limited distribution through specially licensed and closely supervised commercial channels. The brain and medulla must be condemned.

Differential diagnosis : Plant poisoning, botulism, equine infectious anaemia and the dumb form of rabies.

Diseases caused by bacteria

Contagious equine metritis (CEM)

CEM is a contagious, acute venereal disease of horses and other equidae caused by *Haemophilus equigenitalis*.

Transmission : Venereal, contaminated fomites, personnel examining infected animals and rarely stud handlers.

Antemortem findings :

1. Incubation 2 - 10 days after breeding
2. Morbidity up to 100 % from direct contact with an infected stallion
3. Mortality is none
4. Inflammation of the vagina (vaginitis) and copious mucopurulent vaginal discharge (Fig. 184)
5. Hind legs of a mare soiled with exudate
6. No systemic disturbance in affected mares

Postmortem findings :

1. Inflammation of the vagina, cervix and uterus
2. Congestive inflammation of the cervix
3. Mucopurulent exudate in the uterus (Fig. 185) and vagina

Judgement : Carcass of an animal affected with CME is *approved*. If acute inflammation of the vagina and uterus is associated with septicemia, the carcass is *condemned*.

Differential diagnosis : Klebsiella spp. infection, Pseudomonas spp. infection, chronic Staphylococcus infection

Fig. 184: CEM. White, stringy mucous exudate dripping from the vagina of a mare.

Fig. 185: CEM. Accumulation of the mucopurulent exudate in the uterus.

Tetanus

Tetanus is an acute fatal disease of horses caused by *Clostridium tetani*. It is manifested by spasmodic contraction of the voluntary muscles and increased sensitivity to stimuli. Sheep and cattle and rarely pigs are also susceptible.

Transmission : Most frequent transmission of agent in horses is caused by nail wounds. In sheep, the agent may enter after castration and docking; in cows it may enter during puerperal infection, dehorning or castration. In swine, tetanus is mostly seen as a result of wound infection, castration or umbilical infection in new born animals. Clostridium tetani is found in the soil and more commonly in horse manure. It can also be demonstrated in the intestine of healthy horses. Clostridium tetani forms spores which are extremely resistant and may remain viable for years if protected from light and heat. They can however, be destroyed by boiling water. Digestive juices have no effect on spores. Clostridium tetani produces toxins which are responsible for the clinical picture of tetanus. Neuromuscular activity favours migration of tetanus toxins through peripheral nerves which reach the lumbar and cervical region of the cord and the brain stem. In this ascending form of the disease, tetanus develops first in the limbs, followed by the muscles of the trunk. Descending tetanus is observed in horses and humans. Toxins circulate in the blood and lymph and cause tetanus in the muscles of the forelimbs, upper trunk and hind limbs. The first symptoms are the protrusion of the nictitating membrane, and the involvement of facial and jaw muscles leading to lock jaw.

Antemortem findings :

1. Incubation 4 - 14 days to up to 4 months
2. Increased breathing and heart rate
3. Congestion of mucous membranes

4. Stiffness of the masseter muscles and stiff stilted gait
5. Difficulties in mastication of food, hence the common term "lockjaw"
6. Erect ears and prolapse of the third eyelid
7. Animal observed in "sawback stance"
8. Tetany and convulsions
9. Death. At the end of the fatal attack the temperature rises to 42.2°C - 43.3°C. Sheep, goats and swine fall to the ground.

Postmortem findings : No significant postmortem lesions are present.

Judgement : Carcass of an animal affected with tetanus is condemned. The musculature is usually grey yellow in colour and the carcass is inadequately bled.

Differential diagnosis : Strychnine poisoning, hypocalcemia (eclampsia) of mares, cerebrospinal meningitis, lactation tetany of cattle, enzootic muscular dystrophy, enterotoxaemia of lambs, polioencephalomalacia

Glanders

Glanders is a bacterial disease of horses and other solipeds characterized by lesions in lymph glands, lymph vessels, respiratory tract and skin. It is caused by *Actinobacillus (Malleomyces) mallei*.

Transmission : Ingestion of food and drinking of water contaminated with secretions and excretions of sick animals. Wound infection and the respiratory route in acute glanders, contaminated needles, grooming equipment, urine, nasal discharges, purulent skin lesions are also associated with the

transmission of this disease.

Antemortem findings :

Acute form

1. High fever
2. Breathing difficulties and coughing
3. Snoring sound if lesions are in the larynx
4. Ulcers in the nasal mucosa. Star shaped scars upon healing of ulcers
5. Nodules on the skin, abdomen and lower limbs
6. Death due to septicemia

Chronic form

7. Low mortality and high rate of recovered animals
8. Intermittent fever and coughing
9. Unthriftiness and loss of weight
10. Unilateral rhinitis and yellowish-green or bloody nasal discharge
11. Heavy exudate on the skin surface
12. Enlarged submaxillary lymph nodes, abscess formation and abscess rupture.
13. Granulomatous nodules along the lymphatics under the skin, especially on the legs.
14. Swollen pipe like lymphatics (Farcy)
15. Enlarged rear legs

Postmortem findings :

1. Inflammation of the lymph nodes. The nodes are enlarged, fibrotic and abscessed
2. Ulcers in the nasal mucosa, larynx and trachea.
3. Nodules in lungs scattered throughout the lung tissue. These nodules have greyish centres.
4. Nodules on the skin and in the subcutis

5. Necrosis in the internal organs and testicles

Judgement : Carcass of an animal affected with glanders is *condemned*. The animal should not be admitted to the abattoir.

Differential diagnosis : Epizootic lymphangitis, ulcerative lymphangitis, strangles, dourine and melioidosis

Strangles (Distemper)

Strangles is a contagious disease of equines characterized by inflammation of the upper respiratory tract and purulent lesions in the regional lymph nodes. It occurs in stressed young horses 1 - 5 years old. It is caused by *Streptococcus equi*.

Transmission : Source of infection is nasal discharge from infected animals and contaminated food and water. Infection is spread by ingestion or via respiratory route by inhalation of droplets. It may spread for at least 4 weeks after the initial attack due to organism developing resistance to diverse environmental conditions. The spread of infection is also caused by parasites and infected animals during copulation. Infection of the udder of the mare may occur from an infected foal.

Antemortem findings :

1. Incubation 4 - 8 days
2. Temperature 39.5°C - 40.5°C
3. A soft, moist and constant cough
4. Severe pharyngitis and laryngitis
5. "Hot" painful abscesses in submaxillary, pharyngeal and parotid lymph nodes and lymph vessels (Fig. 186)
6. Necrosis of skin and eruption of abscesses

7. Edema of lower limbs (swollen limbs are 3 - 4 normal size)
8. Empyema of the guttural pouch complications
9. Spread of lesions to lungs causing acute pneumonia
10. Purulent inflammation of the brain followed by excitement, neck rigidity and terminal paralysis
11. Pericarditis
12. Lameness and difficult breathing

Atypical form of strangles is manifested by subclinical infection and mild disease.

Postmortem findings :

1. Abscesses in the internal organs including the liver, spleen, lungs etc.
2. Abscesses on the pleura and peritoneum
3. Abscesses in the mesenteric lymph nodes

"Bastard strangles" denotes multiple abscessation in the vital organs and generalized systemic infection.

Fig. 186: Strangles (Distemper). Hot painful abscesses of the lymph nodes of the throat.

Judgement : Carcass of an animal affected with strangles is *condemned* if the animal shows signs of acute infectious disease accompanied with fever and systemic generalized lesions. Atypical, mild form of the disease has a *favourable judgement*.

Differential diagnosis : Epizootic lymphangitis, ulcerative lymphangitis, dourine, melioidosis, equine viral rhinopneumonitis, equine influenza and equine viral enteritis

Parasitic diseases

Diseases caused by protozoa

Trypanosomiasis (Dourine, Mal du coit)

Contagious trypanosomiasis in horses is manifested by edematous swelling and inflammation of the genitalia, cutaneous lesions and paralysis. This disease is caused by flagellated protozoan called *Trypanosoma equiperdum*.

Transmission : Transmission by coitus and rarely by bloodsucking flies (Tabanidae and Stomoxys)

Antemortem findings :

1. Incubation: 1 week to several months
2. Low recurrent fever
3. Loss of condition and anaemia
4. Rough hair coat
5. Acquired loss in cutaneous pigmentation (vitiligo) noted as white discrete patches of various shapes and sizes in the skin and external genitalia.
6. Transitory urticarial plaques which do not ulcerate, appear

on the mucosa and skin, particularly on the flanks.
7. Mucopurulent urethral or vaginal discharge
8. Frequent urination and increased sexual desire
9. Edema of the scrotum, prepuce and penis in stallions and the udder edema in mares
10. Edema of the ventral abdomen
11. Depigmented genital mucosa and rarely ulceration of vaginal mucosa
12. Nervous signs are manifested by incoordination, irregular muscular contractions, facial paralysis and complete paralysis of the body.
13. Clinical signs may be absent in chronic disease.

Postmortem findings :

No specific lesions on postmortem
1. Edema of genitalia
2. Emaciation, anaemia and characteristic depigmentation in the skin and external genitalia
3. Edematous fluid in the pleural, pericardial and peritoneal cavities

Judgement : Carcass of an animal showing chronic lesions of trypanosomiasis without systemic involvement and the carcass of recovered animals is *approved*. Horse carcass affected with the disease is *condemned* if clinical signs are accompanied with emaciation and edema or anaemia.

Differential diagnosis : Equine infectious anaemia

CHAPTER 7

SPECIFIC DISEASES OF POULTRY

Diseases caused by viruses and chlamydia

Avian influenza (Fowl plague, highly pathogenic avian influenza (HPAI))

Avian influenza is a viral disease of several avian species in various parts of the world. The disease can range from asymptomatic and mild to hyperacute and fatal. Avian influenza occurs infrequently in *humans*. It is seen as an occupational hazard, primarily to those associated with varied activities in the poultry industry; employees in abattoirs, vaccinators, laboratory staff and other personnel. In most cases the clinical picture is that of *conjunctivitis* with rare systemic reactions. Avian influenza is reportable disease in many countries. It has to be confirmed by virus isolation.

Transmission : Secretions from infected birds, by wild birds and contaminated feed, equipment and people. Seabirds and migratory waterfowl comprise the main reservoir for avian influenza virus.

Antemortem findings :

1. The incubation period varies from a few hours to about seven days.
2. The morbidity and mortality rates can reach 100 % in cases of highly pathogenic strain of the viruses.
3. Marked depression, loss of appetite and watery diarrhoea
4. Coughing, sneezing, rales, excessive lacrimation

5. Drop in egg production in layers
6. The conjunctiva is congested and swollen, and occasionally haemorrhagic.
7. Swollen combs with cyanotic tips and haemorrhagic surface
8. Edematous wattles (Fig. 187) and edema around the eyes, head and neck
9. Ruffled feathers and dark red skin (Fig. 188)
10. Diffuse haemorrhages between the hocks and feet
11. Blood in the cloaca
12. Some birds may recover, even after being severely affected.

Postmortem findings :

1. Birds that die with the peracute form of AI may show no significant gross lesions
2. Dehydration
3. In highly pathogenic influenza virus, fibrinous exudate is found in airsacs, oviduct, peritoneum and pericardial sacs.

Mild to moderate infection

4. Inflammation of conjunctivae, trachea and airsacs
5. Pronounced congestion of the musculature
6. Ovarian regression in laying birds
7. Edema of the head with congestion, haemorrhages and cyanosis of the combs, wattles and sinuses
8. Vesicles and ulceration of the comb
9. Petechial and ecchymotic haemorrhages in abdominal fat, various serosal and mucosal surfaces, heart, gizzards, proventriculus and small intestine (Fig. 189)
10. The feet often appear edematous with haemorrhages. Red discoloration of the shanks is also noted.

Judgement : Carcasses affected with avian influenza in any form should be *condemned*.

Differential diagnosis : Fowl cholera, chlamydiosis, mycoplasmosis, velogenic viscerotropic Newcastle disease

Fig. 187: AI. Edematous, cyanotic comb and wattles of a chicken.

Fig. 188: Bloody cloaca and dark coloured skin of a chicken that died of AI.

Fig. 189: AI. Haemorrhage in the small intestine, between two dark coloured ceca.

Newcastle Disease (NCD)

Velogenic Viscerotropic Newcastle disease (VVND) or Asiatic Newcastle disease (AND)

NCD is in its chronic form an infection of domestic fowl with symptoms such as rejection of food, listlessness, abnormal breathing, discharge from eyes and greenish diarrhoea. Mortality in chicken is 50 - 80 %, but in adults much lower due to available vaccination. VVND is an acute, fatal infection of birds of all ages with predominant haemorrhagic lesions of the gastrointestinal tract, severe depression, and death prior to clinical manifestations. This disease is caused by the *most virulent strain of the Newcastle disease virus*. The virus of VVND is very resistant and remains viable at extreme pH and temperature ranges, and may remain viable in the bone marrow of poultry carcasses for weeks.

Transmission : Transmission is by direct contact, fomites, and by aerosols through coughing, gasping and respiratory fluids. The virus has a wind borne potential for spread creating quite a

challenge for control and prevention. Faeces and insect and rodent vectors are also involved in the transmission.

Antemortem findings :

1. The incubation period varies from 2 - 15 days.
2. Depression and loss of appetite
3. Sudden death
4. Edema of the head. Swelling of the lower eyelid, often accompanied by conjunctivitis (Fig. 190).
5. Dark ring around the eye (black eye)
6. Excessive fluids from the respiratory tract
7. Paralysed wings and twisting of the head and neck (torticollis)

Postmortem findings :

Acute form

1. Peracute deaths will often show no discernible lesions in some of the first birds dying in an outbreak.
2. The oesophagus shows haemorrhage and erosions.
3. Edema of the head and neck
4. The mucosa of the trachea is frequently haemorrhagic (Fig. 191 upper).
5. Haemorrhages are throughout the gastrointestinal tract with a tendency to ulcerate and become necrotic as the disease progresses.
6. In the intestine there is generally an inflammatory response and marked involvement of the caecal tonsils and Peyer's patches (Fig. 191 middle).
7. The mucosal lining of the proventriculus is a frequent site of haemorrhage, especially at the junction between the oesophagus and proventriculus (Fig. 191 bottom).
8. Edematous or haemorrhagic ovaries
9. In hens that have survived the disease, there is a tendency to

lay misshapen eggs or develop egg yolk peritonitis.

Chronic form

10. Catarrhal inflammation of the respiratory system
11. Edema in surrounding connective tissue

Judgement : Birds with VVND or NCD should not be admitted to the abattoir. If disease is suspected laboratory confirmation should be obtained. If confirmed, carcass is *condemned* and premises with equipment should be disinfected. In case that laboratory confirmation is not possible, suspected carcasses should be also *condemned*. In some countries compensation is paid for condemned birds.

Differential diagnosis : VVND and NCD must be differentiated from the following diseases: Infectious bronchitis, laryngotracheitis, fowl cholera, highly pathogenic avian influenza (HPAI), fowl pox (diphtheritic form), psittacosis, acute Mycoplasma gallisepticum infection, avian encephalomyelitis, vitamin E deficiency, Marek's disease and Pacheco's disease in parrots

Fig. 190: New Castle disease (NCD). Swelling of the lower eyelid and conjunctivitis.

Fig. 191: NCD. Acute form: Haemorrhage in the mucosa of the trachea (upper), large intestine, particularly caecal tonsils (middle), proventriculus (bottom) and gizzard.

Infectious bronchitis (IB)

Infectious bronchitis is an acute, highly contagious viral disease of chickens, manifested by respiratory signs, renal disease and a significant drop in egg production.

Transmission : Airborne transmission in the direction of prevailing wind. The spread of infection is rapid in a flock. Some birds become carriers and shedders of the virus through secretions and discharges for many months after the infection. IB virus persists in contaminated chicken houses for approximately four weeks.

Antemortem findings:

1. Indifference and depression
2. Sneezing, gasping and coughing
3. Nasal discharge
4. Abnormal respiratory sounds (rales)

5. Weakness and huddling near the light source
6. Reduced egg production in laying birds. Low egg quality and soft egg shells are noted.
7. Mortality due to kidney disease caused by the nephrotropic strain of the IB virus.
8. Inflammation of the air sacs may be a complication of IB.

Postmortem findings :

1. Serous, catarrhal and caseous exudate in the upper respiratory tract including nasal passages, trachea, sinuses and bronchi
2. Cloudy airsacs
3. Abdominal airsacs may contain yellow caseous exudate (Fig. 192).
4. Occasionally swollen and pale kidneys containing urolith deposits (uric acid crystals) (Fig. 193)
5. Yolk material or fully formed egg in the abdominal cavity in layer hens
6. Small cystic oviducts

Judgement : Affected birds are treated as suspects on antemortem inspection. A carcass showing acute signs of clinical disease accompanied with emaciation is *condemned*. A carcass in good flesh and without systemic changes is *approved*. The affected parts are *condemned*.

Differential diagnosis : Newcastle disease, laryngotracheitis (LT) and infectious coryza. Laryngotracheitis spreads slowly in a flock although respiratory signs are more severe than in infectious bronchitis. LT is not seen in young chicken.

Fig. 192: IB. Abdominal airsac containing yellowish caseous exudate.

Fig. 193: IB. Swollen kidneys and ureters containing urolith deposits (uric acid crystals).

Laryngotracheitis (LT)

LT is an acute viral disease of chicken characterized by difficult breathing, gasping and coughing up of bloody exudate.

Transmission : Virus entry in LT is via the respiratory route and

the intraocular route. Oral infection may also occur. The transmission from acutely infected birds is more common than from recovered or vaccinated birds. The latter may shed the virus for a prolonged period of time. Mechanical transmission via fomites is another possibility.

Antemortem findings :

1. Incubation 6 - 12 days following natural exposure
2. High morbidity and moderate mortality (10 - 20 %)
3. Difficult breathing and coughing (Fig. 194)
4. Loud gasping or wheezing sounds
5. In mild form of LT, lacrimation, nasal discharge, swelling of conjunctivae and sinuses and reduced egg production.

Most chicken usually recover in 10 - 14 days and up to 4 weeks in severe cases.

Postmortem findings :

1. Inflammation of the larynx and trachea leading to necrosis and haemorrhage of mucosa
2. Extension of infection to the bronchi, lungs and airsacs
3. Death due to pseudomembranes or cheesy plugs in the trachea (Fig. 195)

Judgement : Mild form of disease and recovered birds may have a *favourable judgement* if carcass is in good flesh. If an acute condition is associated with general systemic changes, the carcass is *condemned.*

Differential diagnosis : Newcastle disease, Infectious bronchitis and infectious coryza

Fig. 194: LT. Difficult breathing and coughing.

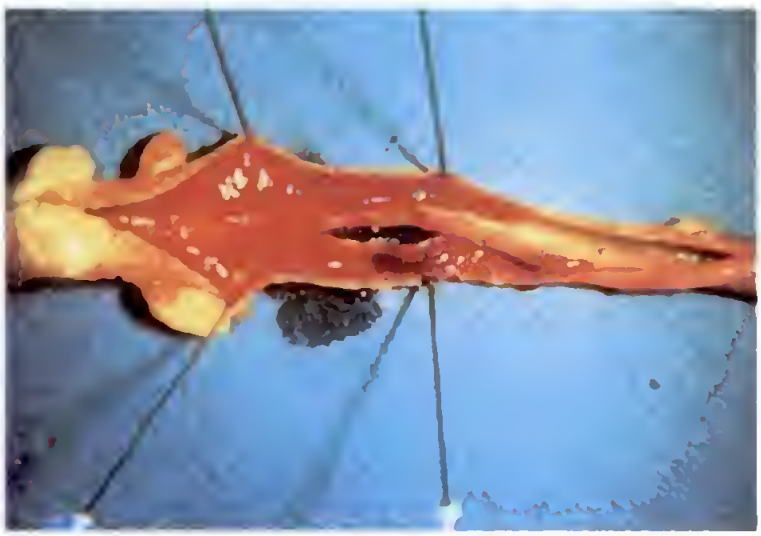


Fig. 195: LT. Inflamed trachea containing cheesy plugs.

Fowl Pox (FP, Pox, Avian Pox)

Fowl Pox is a viral disease of chicken, turkeys and other birds distinguished by cutaneous lesions on the head, neck, legs and feet. It has a worldwide distribution and affects birds of all age groups, except the recently hatched.

Transmission : The virus is present in lesions and in desquamated scabs. It is resistant to environmental factors and persists in the environment for many months. It usually infects birds through minor abrasions. Mechanical transmission occurs by cannibalism. Some mosquitoes can transmit the virus from infected to uninfected birds. The virus can be also transmitted by injury to the skin.

Antemortem findings : Two forms of lesions are recognized, - the cutaneous (dry form) and the diphtheric (wet form)

Cutaneous form

1. Low mortality
2. Lack of flock vigour and weight loss
3. A mild to moderate loss in egg production
4. Scabby lesions on the head, neck and unfeathered parts of the skin (Fig. 196)

Diphtheric form

5. Mortality low to moderate
6. Difficult breathing
7. Nasal and ocular discharge

Postmortem findings : The following stages of the pox lesions *papules, vesicles* and *pustules* may be observed.

Cutaneous lesions

1. *Papules* are light coloured nodules.
2. Vesicles and *pustules* are raised and commonly yellow.

Diphtheric lesions

3. Buff to yellow plaques on mucous membranes in the mouth (Fig. 197), oesophagus and upper respiratory tract
4. Occlusion of trachea, and death due to asphyxiation

Histopathology shows characteristic intracytoplasmic inclusion bodies (Bollinger bodies) in the infected epithelium.

Judgement : Carcass affected with fowl pox is *condemned* if progressive generalized lesions in a bird are accompanied with emaciation. Fowls with localized lesions and recovered birds are *approved* after the removal of scales.

Differential diagnosis : Pantothenic acid and biotin deficiency, vitamin E deficiency, infectious laryngotracheitis and other respiratory diseases in poultry, injuries caused by external parasites and cannibalism.

Fig. 196: FP. Cutaneous form (dry pox). Face lesions in young turkey.

Fig. 197: FP. Diphtheric form (wet pox). Caseous lesions in the mouth and throat of a chicken.

Avian leucosis complex

Avian leucosis complex occurs in four separate disease entities:

1. Leucosis-sarcoma group (Lymphoid leukosis)
2. Marek's disease (MD)
3. Reticuloendotheliosis group (REV)
4. Lymphoproliferative agent of turkey

Transmission : L/S virus is transmitted by egg in vertical transmission and by shedders in horizontal transmission (chicken to chicken). Tumour lesions may or may not develop. Developed tumours can be differentiated by the bird's age on necropsy, histology and impression smears. In horizontal transmission, chicken which contract the virus after hatching develop antibodies; some will remain shedders, some will develop tumours and die, and others will overcome the infection. Infection from flock to flock is unlikely as the virus does not survive a long time in the environment. In many chickens the virus may be also in a latent state.

(A) Lymphoid leucosis (big liver disease, visceral lymphomatosis)

This disease is being studied because of its economic significance, and also as an experimental model of cancer. Lymphoid leucosis is a B cell tumour which starts in the bursa and, before sexual maturity, may spread to other organs. Mature birds are more affected than young birds. Male birds are also affected in lesser numbers than female due to the earlier regression of bursa in male birds.

Antemortem findings :

1. The disease occurs in 14 - 30 weeks old birds.
2. Pale, shrivelled comb and loss of appetite
3. Dehydration and emaciation

4. Diarrhoea, green scant faeces
5. Enlarged liver, bursa of Fabricius and kidneys
6. Distended abdomen due to enlarged liver
7. Reduced egg production

Postmortem findings :

1. Grey tumour lesions[4] in the liver (Fig. 198), spleen and bursa
2. Other organs such as lung, heart, proventriculus, gonads, bone marrow and mesentery are sometimes affected.
3. Ecchymotic haemorrhages around the skin follicles of the wing

Judgement : The carcass of a bird affected with lymphoid leucosis is condemned. The condemned material may be used for animal food.

Differential diagnosis : Between lymphoid leucosis and Marek's disease is shown in Table 3.

Fig. 198: Avian leucosis complex. Diffuse nodular lesions in the liver, spleen, intestine and heart.

[4] Tumour lesions are nodular, miliary or diffuse. Nodules are approximately 0.5 cm - 5 cm in diameter, miliary lesions are 2 mm in diameter and diffuse lesions are noted as a uniform enlargement of the organs.

(B) Marek's Disease (Polyneuritis, Fowl paralysis, Neurolymphomatosis gallinarum)

Marek's disease is caused by the *herpes virus* (DNA).

Transmission : It is spread by airborne infection involving follicle cells called chicken dander. Transmission of the virus is horizontal. At room temperature the virus of Marek's disease remains viable for 16 weeks and in litter for 6 weeks. Birds are most susceptible to the infection during the first few weeks of life. Infected birds will start to shed the virus in the second or third week after infection and will continue to do so throughout their life, although they do develop antibodies against the virus.

Marek's disease is commonly associated with coccidiosis in the field. This is probably due to lack of immunity against coccidiosis in birds affected with Marek's. There are 6 types of Marek's disease.

1. Peracute in 3 - 5 weeks old chicks; manifested with sudden death
2. Anaemia in 3 - 6 weeks old chicken
3. Classical Marek's disease (range paralysis) showing paralysis of two wings and legs due to peripheral nerve involvement. The nerves are 2 - 3 times their normal size. Central nerve involvement may also occur.
4. Acute Marek's disease occurs mainly in 6 - 12 weeks old birds. It is manifested with tumours in the liver, spleen, kidneys, brain, spinal cord and dorsal root ganglia and with sudden death. The heart, lungs, gonads and muscles may also be involved.
5. Skin leucosis. Noted in broiler chicken on postmortem examination in abattoirs as enlargement of feather follicles and associated lymphoid infiltrations (Fig. 199).
6. Transient paralysis in 12 - 18 weeks old pullets become

paralysed and may recover with 24 hours if moved to a quiet place.

Fig. 199: Avian leucosis complex. Neoplastic lesions of Marek's disease in the leg.

Postmortem findings :

1. Skin neoplasia
2. Enlarged spleen
3. Diffuse proliferation of white pulp

Judgement : Carcass of a chicken affected with Marek's disease is *condemned* in the extensive cutaneous form and in the visceral form. Localized skin lesions require the *condemnation* of the affected portions, the rest of the carcass may be approved. The condemned material may be used for animal food.

Differential diagnosis (for skin lesions): Lymphoid leucosis (see Table 7), erythema, dermatitis, pigmentation and normal large follicles. If the central nervous system is affected the lesions of Marek's disease should be distinguished from those of Newcastle disease and of avian encephalomyelitis. In immunized birds for Marek's disease, the inflammation of the feather follicles (folliculitis) may be caused by the Marek's disease virus. In both cases follicular enlargement is noted; however, the lesion may

differ in colour. In immunized birds the lesions of folliculitis are yellowish-white, whereas with the Marek's virus, lesions are blue-grey in colour with a pinkish appearance due to petechial haemorrhage.

Table 3: Differential diagnosis between lymphoid leucosis and Marek's disease

Feature	Lymphoid leucosis	Marek's diseases
Age of onset	16 weeks	4-6 weeks or older
Symptoms	Absent	Frequently paralysis or paresis
Incidence	Seldom above 5 %	Usually above 5 %
Gross Lesions		
Peripheral nerve enlargement	Absent	Usually present
Bursa of Fabricius	Nodular tumours	Diffuse enlargement or atrophy
Skin, muscle or proventriculus tumours	Usually absent	May be present
Microscopic Lesions		
Peripheral nerve infiltration	Absent	Present
Cuffing in white matter of cerebellum	Absent	Present
Tumour in the liver	Focal or diffuse	Frequently perivascular
Bursa of Fabricius	Intra-follicular tumour	Inter-follicular tumours or atrophy
Follicular patterns of lymphoid cells infiltration in the skin	Absent	Present
Cytology	Uniform lymphoblasts	Pleomorphic mature and immature cells including lymphoblast, small medium and large lymphocytes and reticulum cells.

Ornithosis (Psittacosis, Avian chlamydiosis)

Ornithosis is an acute or chronic disease of turkeys, ducks, chicken, pheasants and pigeons. It is caused by *Chlamydia psittaci*. In psittacine birds such as parrots, parakeets, cockatoos, macaws etc. and humans, this disease is called psittacosis.

Transmission : Wild carrier birds and cage birds transmit Chlamydia to their nestling which may survive and become carriers. Carrier birds shed Chlamydia in their secretions and excretions. Chlamydia present in faecal dust may be inhaled or ingested. Pigeons are suspected of being disseminators of infection.

Antemortem findings :

1. Mild cases may be unobserved or show mild respiratory signs and diarrhoea

In turkeys

2. Depression and weakness
3. Reduced weight
4. Nasal discharge
5. Green yellow diarrhoea
6. Watery diarrhoea in ducks, geese

In pigeons

7. Unilateral or bilateral conjunctivitis
8. Depression and loss of appetite
9. Abnormal respiratory sounds (rales) and watery diarrhoea

Postmortem findings :

1. Inflammation of the lungs, airsacs, liver, heart, spleen, kidneys and peritoneum

In turkeys

2. Wasting
3. Vascular congestion
4. Fibrinous inflammation of pericardium, airsacs, lungs
5. Congestion of the lungs and an enlarged congested spleen

In pigeons

6. Swollen eyelids and conjunctivitis
7. In cage birds enlarged spleen and liver, inflammation of the airsacs and pericardium and intestinal congestion.

Judgement : If the disease is suspected on antemortem, birds are treated as suspects and samples should be shipped to the Laboratory. All reasonable precautions should be taken to avoid risk of human contact. If the disease is suspected on postmortem, delayed slaughter of the birds from the same source should be required. Samples are also forwarded to the diagnostic laboratory. Positive laboratory finding necessitate *condemnation* of the bird or carcass. If the disease is not confirmed the carcass may be approved if otherwise wholesome.

Differential diagnosis : Mycoplasma gallisepticum infection, pasteurellosis, salmonellosis (pullorum disease; bacillary white diarrhoea(BWD))

Diseases caused by bacteria

Salmonellosis

Salmonellosis is responsible for significant losses to the poultry industry. Salmonella infections in this Manual include *pullorum disease, fowl typhoid, arizona infection* and *paratyphoid*. Pullorum disease occurs in chicken and turkeys and is caused by *Salmonella*

pullorum. The greatest losses are in chicken less than 4 weeks old.

Antemortem findings (in young chicks) :

1. Anorexia
2. Diarrhoea
3. Pasting of vent
4. Stunted, unthrifty
5. Poor feathering
6. Pale shrunken comb and wattles

Postmortem findings :

1. Multiple grey nodules in the heart (Fig. 200), lungs, liver, spleen, peritoneum, the gizzard, intestine and pancreas of a poult
2. Abnormal ovary showing discoloured follicles in an adult bird
3. Rarely peritonitis, ascites or oviduct impaction
4. Swelling of the tibiotarsal joint

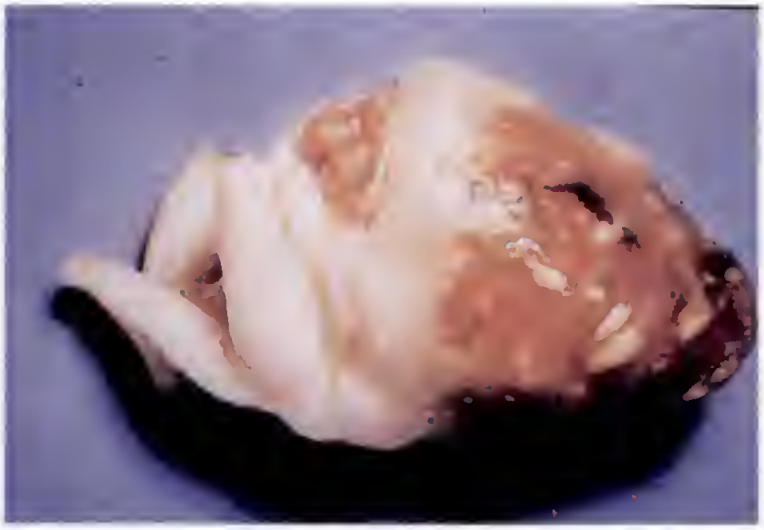


Fig. 200: Pullorum disease. Multiple grey nodules in the heart.

Judgement : Carcass and viscera affected with pullorum disease are *condemned.*

Differential diagnosis : Liver and heart lesions should be differentiated from infections due to other salmonellae and from campylobacteriosis, colibacillosis and omphalitis. Nervous lesions should be distinguished from nervous signs observed in Newcastle disease. Respiratory tract lesions should be differentiated from aspergillosis and joint lesions with synovitis and bursitis caused by other bacteria or viruses.

Fowl typhoid

Fowl typhoid is an infectious disease in chicken and turkeys and rarely in other poultry, game birds and wild birds. The causative agent is *Salmonella gallinarum.* It is seen more often in adult birds. Antemortem signs of fowl typhoid and pullorum disease are similar in birds.

Postmortem findings :

1. Enlarged, bronzed liver and enlarged spleen in a turkey (Fig. 201).
2. Enlarged kidneys
3. Pale cadaver
4. Inflammation of the anterior part of the intestine

Judgement : Carcass and viscera affected with fowl typhoid are *condemned.*

Differential diagnosis : see pullorum disease

Fig. 201: Fowl typhoid. Enlarged, bronzed to the mahogany colour liver and enlarged, mottled and brittle spleen in a turkey Diseased liver and spleen are shown in contrast to the normal ones at left.

Paratyphoid infection

Paratyphoid infection is an acute and chronic infection of poultry and mammals. Ten to twelve species of *Salmonella* are associated with most outbreaks in poults. The most commonly involved is *S. typhimurium* in birds less than one month old.

Antemortem findings :

1. Drooping wings
2. Shivering and huddling near a heat source
3. Muscular incoordination and trembling

Postmortem findings :

1. Nodular lesions in the pancreas
2. Button type lesion in the intestine (Fig. 202)
3. Enteritis
4. Dehydration
5. Unabsorbed yolk material and omphalitis
6. Less frequent are joint infections
7. Blindness
8. Swollen eyelids

Judgement : Carcass and viscera affected with paratyphoid infection are *condemned*.

Differential diagnosis : see pullorum disease

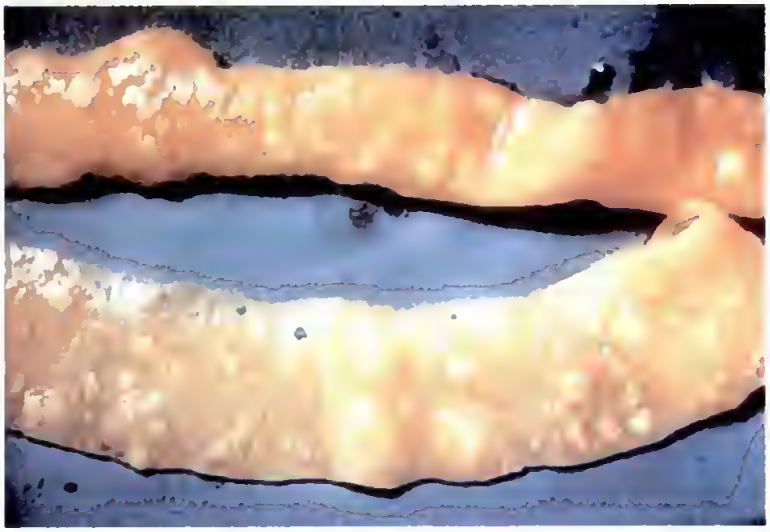


Fig. 202: Paratyphoid infection. Button type lesion in the intestine.

Arizona infection (Arizoonosis, Paracolon infection)

Arizoonosis occurs in young turkey poults. It is caused by Arizona hinshawii (Syn. Salmonella arizona). Arizona infection is egg transmitted.

Antemortem findings :

1. Listlessness and trembling
2. Pasting of vent area with faeces
3. Huddling near a light source
4. Twisted head and neck
5. Cloudiness and enlargement of the eye causing blindness

Antemortem findings : Antemortem findings in young birds are similar to those of paratyphoid.

Postmortem findings :

1. Enlarged yellow liver
2. Congestion of the duodenum
3. Unabsorbed yolk material
4. Cheesy plugs in the intestine or caecum
5. Inflammation of the oviduct and peritoneum
6. Eye lesions
7. Purulent exudate in the brain

Judgement : Carcass and viscera affected with arizoonosis are *condemned*.

Differential diagnosis : The causative organism must be isolated and identified for differentiation from salmonellosis.

Fowl cholera (Pasteurellosis)

Fowl cholera is an infectious disease affecting almost all classes of fowl and other poultry. The disease is more prevalent in turkeys than in chicken. It occurs more frequently in stressed birds associated with parasitism, malnutrition, poor sanitation and other

conditions. Fowl cholera is caused by *Pasteurella multocida*. This organism is easily destroyed by sunlight, heat, drying and most of the disinfectants. However, it will survive several days of storage or transportation in a humid environment. It persists for months in decaying carcasses and in moist soil. The agent is frequently carried in the oral cavity of wild and domestic animals.

Transmission : If birds are bitten by infected animals such as rodents and carnivores, the disease could be disseminated in the flock. Contaminated feed, water, soil and equipment are also considered as potential factors in the spreading of the disease.

Antemortem findings :

Acute septicemic form

1. High morbidity and mortality and sudden death
2. Dead birds may be found on dropping boards or in nests.
3. Depressed, cyanotic and loss of appetite
4. Nasal and oral discharge
5. Greenish diarrhoea
6. The comb may be swollen and discoloured.
7. Emaciated

Chronic fowl cholera

8. Swelling of wattles, sinuses, joints, foot pad and tendon sheaths
9. Cheesy exudate in the conjunctival sac
10. Twisting of the head and neck may be observed in some birds.
11. Middle ear infection is rare but occurs when the bacterial agent reaches the middle ear through the nasal cavity.
12. The bird may lose its sense of balance with the head and neck twisted to one side.
13. If both ears are infected, the bird's head and neck are pulled back over the body and between the legs.

Postmortem findings : In the very acute stages, lesions may be lacking.

1. Caseous exudate in wattles, sinuses (Fig. 203), the nasal turbinates, middle ear, joints or tendon sheaths.
2. Petechial and ecchymotic haemorrhages on the heart, serous and mucous membranes, on the gizzard and abdominal fat.
3. The liver is swollen and is streaked with white areas and associated small grey areas of necrosis (corn meal liver, Fig. 204).
4. Free yolk in the peritoneal cavity in breeder hens and layers.
5. Acute oophoritis and peritonitis are often seen.
6. The lining of the upper intestine is reddened and gut content is slimy.

In chronic cases

7. Darkened breast muscle is frequently noted and haemorrhagic lesions are often missing.

Judgement : Localized lesions of pasteurellosis such as infection of wattles, joints or tendon sheaths require the *condemnation* of the affected parts; the rest of the carcass is approved. Septicemic carcasses should be *condemned.*

Differential diagnosis : Acute colibacillosis and erysipelas in turkeys, salmonellosis, tuberculosis, listeriosis. Pasteurellosis is differentiated from septicemic and viremic diseases by culture of P. multocida. Closely related bacteria such as S. gallinarum, P. haemolytica and others may cause a cholera like disease or they may complicate other diseases.

Public health significance : Fowl cholera, although known for 200 years is still a poorly controlled disease in birds. Humans may become infected with this disease, and then they may also infect poultry with exudates from the nose and mouth.

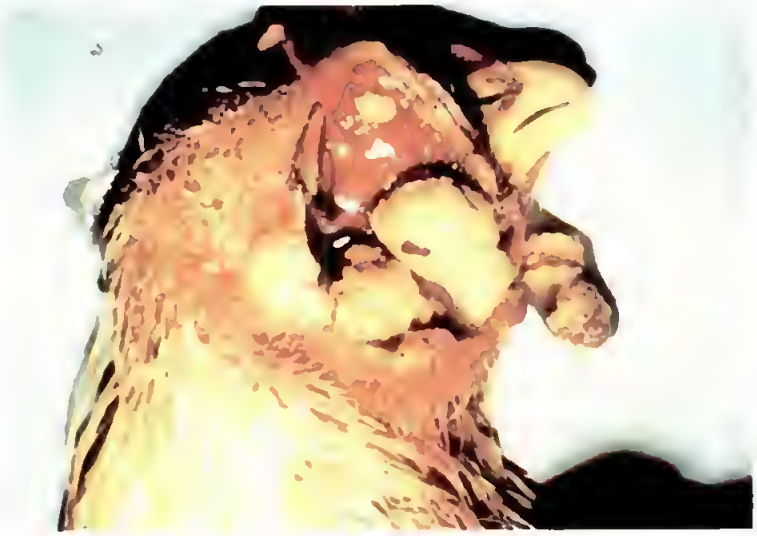


Fig. 203: Fowl cholera. Cheesy exudate in the infraorbital sinus.

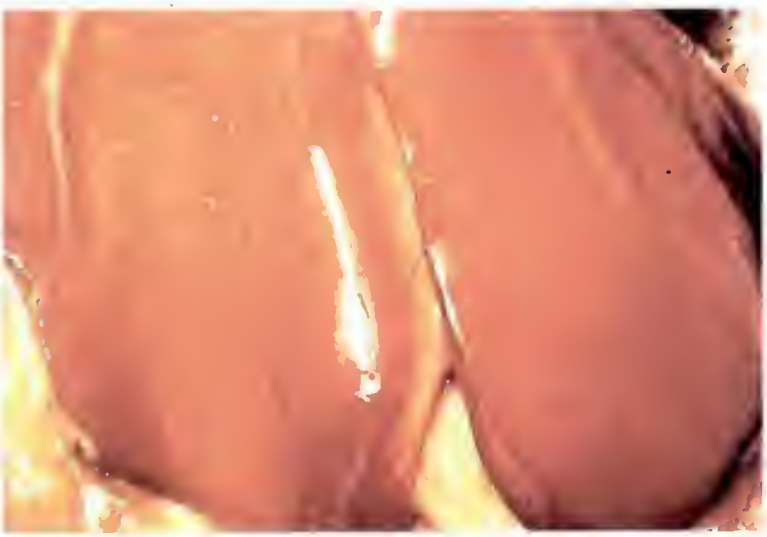


Fig. 204: Fowl cholera. Small areas of necrosis in the liver (corn meal liver).

Tuberculosis

Tuberculosis is a chronic granulomatous disease of poultry caused by *Mycobacterium avium*. It is usually found in older chicken which are kept beyond the laying season. Other poultry are also affected.

Antemortem findings :

1. Depressed and progressive loss of weight leading to emaciation
2. Pale skin of the face, wattles and comb
3. Dull, ruffled feathers
4. Diarrhoea
5. Unilateral lameness

Postmortem findings :

1. Irregular greyish yellow or greyish white nodules of different sizes in the liver (Fig. 205), spleen, and intestine
2. In advanced cases, TB nodules are found in the bone marrow.
3. Lesions are rarely found in the heart, ovaries, testes and skin.
4. TB lesions on organ surfaces are easy enucleated from the surrounding tissue. Mineralization is not present in the lesions.

Judgement : A carcass affected with tuberculosis is condemned.

Differential diagnosis (on postmortem) : Aspergillosis, typhoid and paratyphoid, salmonellosis, fowl cholera, campylobacter, colibacillosis, chlamydiosis, histomoniasis and neoplasm. In neoplasm, the cut surface of the lesion is uniform and without necrosis.

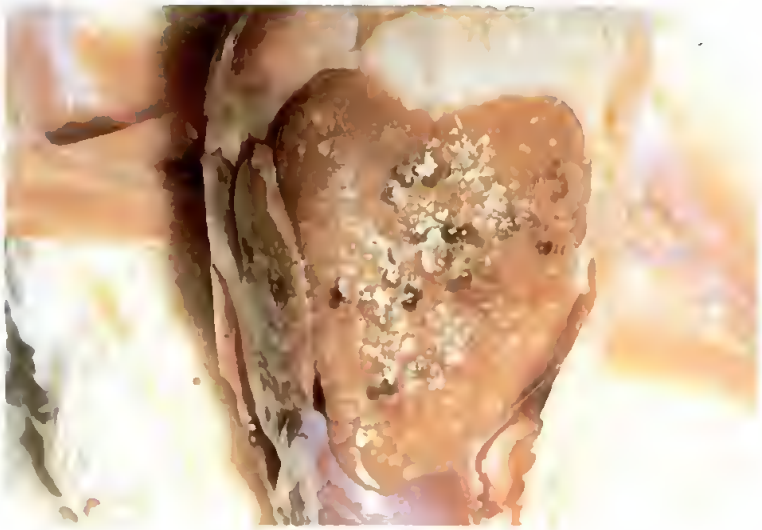


Fig. 205: Tuberculosis. Greyish white nodules in the liver.

"Air sac disease" and Chronic Respiratory Disease (CRD)

The term "Air sac disease" usually refers to a respiratory syndrome characterized by airsacculitis, perihepatitis and pericarditis in broiler chickens between 4 - 8 weeks of age. Pneumonia is also frequently present. Primary factors associated with the etiology of air sac disease are poor air quality and dust, associated with either *viral* or *mycoplasmal agents*. *E. coli* is usually a secondary invader.

Chronic respiratory disease (CRD) refers to respiratory infection of the upper respiratory tract of chicken caused by *Mycoplasma gallisepticum*. This agent affects turkeys more severely and causes infectious sinusitis.

Postmortem findings : Postmortem examination of affected chicken reveals inflammation of trachea and frothy exudate in the air sacs. With presence of secondary invaders, inflamed airsacs have a opaque appearance in the early stages of infection (Fig. 206). In the later stages airsacs are thickened and caseous yellow

exudate is usually present. Yellow fibrinous deposits on the pericardium (pericarditis) and liver (perihepatitis, Fig. 207) are also observed.

Fig. 206: Chronic Respiratory Disease. Cloudy appearance of the abdominal airsacs in this 7 week old chicken.

Fig. 207: Pericarditis and perihepatitis in an eight week old chicken. E. coli was isolated from both lesions.

Judgement : In poultry inspection, it is of great importance to differentiate between the inflammation of the air sacs (*airsacculitis*) and *peritonitis*, as well as pathologic changes in the air sacs and bones. The communication of some air sacs and bones has to be observed during the judgement of carcasses with

airsacculitis and bone diseases. Birds affected with airsacculitis are treated as suspects on antemortem examination. On postmortem examination affected parts with localized lesions are *condemned* and the rest of the carcass is *approved* if in good condition and no spread of infection into bones is noted. The rational for approving a carcass in good condition with localized lesions of airsacculitis may be supported with frequent negative findings of microorganisms in these lesions. Since an accumulation of the fluid in the airsacs and associated opacity may be early signs of infection, the other birds in the flock must also be examined. Sufficient evidence must be obtained in that these lesions would remain localized. Otherwise the carcass must be *condemned*. Generalized and extensive lesions of airsacculitis require *total condemnation* of the carcass. If pericarditis or perihepatitis are the only lesions present on postmortem the carcass may be approved if otherwise wholesome.

Parasitic diseases

Diseases caused by protozoa

Histomoniasis (Enterohepatitis or Blackhead)

Histomoniasis is a protozoal disease of turkeys, chicken and game birds. It often occurs in turkeys run with or after chicken. It is caused by *Histomonas meleagridis* which is transmitted in the ova of caecal worms (Heterakis gallinarum) and by earth worms. Well developed hepatic and caecal lesions are pathognomonic for histomoniasis.

Antemortem findings :

1. In young turkeys, mortality and morbidity may reach 100 %.
2. Indifference and loss of appetite
3. Depression, drooping wings and closed eyes
4. Cyanotic (black) head
5. Yellow sulphur coloured faeces
6. Blood in faeces
7. Emaciation in older birds

Postmortem findings :

1. The ceca are characterized with a thickened wall, ulcerated mucosa and caseous exudate.
2. If the caecal wall becomes perforated, inflammation of the peritoneum is observed.
3. The liver lesions consist of circular yellow necrotic tissue surrounded with white rings (Fig. 208).

On microscopy, histomonas are found in the caecal wall or in the liver lesion.

Fig. 208: Histomoniasis. Circular yellow necrotic tissue surrounded with white rings of a turkey liver.

Judgement : The bird is *approved* if in good flesh. The liver and intestine are *condemned*. The carcass is *condemned* if emaciated or associated with septicemia.

Differential diagnosis : Salmonellosis, coccidiosis. aspergillosis and trichomoniasis.

Coccidiosis

Coccidiosis is a major disease problem in commercial poultry and the most common cause of enteritis. It is caused by various species of the protozoan genus Eimeria. At least nine species have been described in chicken. The most important of these are *E. acervulina, E. brunetti, E. maxima, E. necatrix* and *E. tenella.*

The identification of different species of coccidia is based on the distribution of the lesion in the intestine, gross and microscopic appearance, and the size and shape of oocysts. Coccidia are species specific which means that coccidia in chicken will not be found in turkeys and vice versa.

Whether the coccidia actually cause illness or death of the bird depends on the species, dose, location of parasitic reproduction and immune status of the bird. Birds affected with Marek's disease and IBD are more susceptible to coccidiosis.

If parasitic reproduction occurs in the epithelium of the intestine, disease follows and if it occurs subendothelially, the birds usually bleed to death in acute cases.

With ingested *oocysts* moving to the intestinal lining, the life cycle of coccidia begins. Intestinal cells are damaged and in 4 - 8 days, depending on the species, oocysts are shed in the faeces.

Within 12 - 30 hours in a moist environment and in a temperature of 20 - 30°C, the oocysts sporulate and become infective. Birds raised on the range will have more outbreaks of this condition in the spring and summer compared to birds raised in total confinement of barnyards. In the latter, outbreaks can occur at any time.

Coccidiosis causes severe losses in intensive broiler production and in layers, it causes a drop in egg production. This is in spite of great discoveries of many different coccidiostats. Even when the disease is controlled, medication expenses run very high.

Antemortem findings :

1. Ruffled feathers
2. Anaemia and dehydration
3. Retracted head and neck
4. Bloody diarrhoea
5. Weight loss

Findings in poults include:

6. Listlessness and droopiness
7. Ruffled feathers
8. Light brown mucoid diarrhoea
9. Huddling in groups

Judgement : Birds affected with severe coccidiosis associated with emaciation or anaemia are *condemned*. Otherwise, the carcass is *approved*, after the *condemnation* of affected tissue.

Differential diagnosis (on postmortem) : Necrotic enteritis, ulcerative enteritis, salmonellosis, ascariasis, capillariasis, haemorrhagic disease, leucosis, blackhead, bluecomb and haemorrhagic enteritis in turkeys

I. Chicken coccidiosis

(A) E. acervulina

E. acervulina is found in the duodenum and upper jejunum. It is frequent in growing birds and broilers and causes duodenal coccidiosis.

Postmortem findings :

1. Whitish transverse lesions which may coalesce.
2. Parasite location in tissue section is epithelial.

(B) E. brunetti

E. brunetti is found in the lower small intestine, rectum and proximal part of ceca.

Postmortem findings :

1. Fibrinous inflammation of the intestine (fibrinous enteritis) with bloody mucoid exudate
2. Whitish blood streaked faeces

(C) E. maxima

E. maxima is found in the middle part of the small intestine and less frequently, throughout the intestine

Postmortem findings :

1. Haemorrhagic enteritis showing a thickened wall and petechial haemorrhage
2. Large oocysts on microscopy

(D) E. tenella

E. tenella is found in the ceca. It causes caecal coccidiosis.

Postmortem findings :

1. Haemorrhage in the ceca (Fig. 209)
2. In later stages core, of clotted blood and thickened whitish mucosa
3. In young birds, bloody diarrhoea

(E) E. necatrix

E. necatrix is found in the middle part of the intestine near the yolk sac diverticulum. In severe cases, this coccidium is observed throughout the intestine and ceca. It causes acute mortality and weight loss in laying birds.

Postmortem findings : Intestinal distention, mucoid blood filled exudate and white spots noted on the serosa (Fig. 210)

Fig. 209: Coccidiosis. Haemorrhage in the ceca characteristic of E. tenella infection.

Fig. 210: Intestinal distention, mucoid blood filled exudate and white spots noted on the serosa with E. necatrix infection.

II. Coccidiosis in turkeys

Three species of coccidia in turkeys are considered pathogens. These include *E. meleagrimitis, E. adenoeides* and *E. gallopavonis*.

(A) E. meleagrimitis

E. meleagrimitis is found in small intestine. It is the most pathogenic for turkeys.

Antemortem and postmortem findings :

1. Dehydrated birds showing the breast skin adhering to underlying muscles
2. Severe inflammation in duodenum and upper jejunum
3. The upper part of intestine contain excessive fluid and the lower part contain a greenish mucus

(B) E. adenoeides

E. adenoeides is found in the lower small intestine, ceca and large intestine.

Postmortem findings :

1. Severe inflammation of intestinal mucosa and swollen and edematous intestinal wall
2. Fluid faeces with mucus and small amount of blood

(C) E. gallopavonis

E. gallopavonis is found in the lower one-third of small intestine, large intestine and ceca.

Postmortem findings :

1. Necrotic enteritis associated with creamy white exudate
2. Thickened and damaged intestinal wall devoid of food

CHAPTER 8

SPECIFIC DISEASES OF RABBITS

Diseases caused by bacteria

Pasteurellosis (Snuffles, pneumonia)

The Pasteurella species cause various diseases in rabbits. The most common organisms are Pasteurella haemolytica and Pasteurella multocida. *Pasteurella haemolytica* infection in rabbits is associated with chronic rhinitis (colds), pneumonia or death. *Pasteurella multocida* causes chronic rhinitis, colds, snuffles, pneumonia, peritonitis and septicemia.

Transmission : *Pasteurella organisms* are found in the environment and in mucous membranes of healthy animals particularly in the respiratory tract mucosa. There are some predisposing factors involved with susceptibility of Pasteurella infection in rabbits including avitaminosis, inadequate diet, poor husbandry and hygiene, fungal and parasitic infections. The most common source of infection is contact with other infected rabbits and usually, an animal is infected through the respiratory tract. The organism can also be spread from contaminated equipment and cages.

Antemortem findings :

Chronic rhinitis (Colds):

1. Sneezing
2. Clear, watery or thick-yellow nasal discharge
3. Soiled discoloured fur on the inner side of the front legs
4. Muzzle covered with discharge

Rabbits usually do not recover from this infection and may have periodic flare-ups of the cold. The colds may lead to classically described snuffles and further to pneumonia.

Snuffles (Contagious catarrh): This is a chronic and destructive form of cold in rabbits.

1. Frequent "snuffles" and forceful loud sneezing in rabbits
2. Mucoid to cream coloured purulent nasal discharge
3. Purulent conjunctivitis and cloudy eyes
4. Death due to weakness and secondary pneumonia or septicemic infection

Pneumonia:

1. Elevated temperature
2. Dullness and noisy forceful breathing
3. Bluish ears and eyes
4. Death anywhere from 12 hours to 4 days. The survivors may be stunted.

Peritonitis:

1. High temperature
2. Fast and shallow breathing
3. Reluctance to move due to sore abdomen

Septicemia:

1. A dead rabbit may be the first sign
2. Extreme weakness and high temperature
3. Difficult (heavy) breathing
4. Bluish discoloration of the ears and skin
5. Abortion in breeding does

Abscesses:

1. Abscesses on the neck, dewlaps, ribs and back
2. Abscesses in the mammary gland of a doe

Eye and middle ear infection:
1. Partial or complete blindness
2. Pronounced head tilt (may fall over easily)
3. Inability to right themselves

Mastitis: Swollen, bluish glands in lactating does. Ulceration and sloughing may occur with discharge of pus from diseased tissue.

Metritis:
1. White discharge from the vulva
2. Abortion with poor breeding success

Arthritis: Enlarged, painful swollen joints

Postmortem findings :

Snuffles: Inflammation and necrosis of nasal passages which contain mucoid to white purulent material

Pneumonia:
1. Consolidated inflamed area in the lungs. Deep red, sharply demarcated lung lesion and whitish purulent material in the bronchi
2. Cheesy material (fibrin) on the pleura
3. Inflammation of the pericardium and trachea
4. Death caused by inflammation of pleura and collapsed lungs

Peritonitis:
1. Yellow-white deposits (fibrin) on the peritoneum and abdominal cavity (Fig. 211)
2. Abdominal organs adherent to the peritoneum and with one another

Septicemia:
1. Haemorrhages on body fat and heart muscles

2. Enlarged body organs
3. Bluish discoloration of body tissues

Abscesses: Walled off abscesses containing white creamy cheesy pus.

Eye and middle ear infection:
1. Normal eye structure is obliterated by white or yellow puss
2. White pus in one or both middle ears with rare extension to the brain

Mastitis: Swollen mammary gland with red to blue discoloration and congestion. White abscesses may be observed in the gland.

Metritis: Distended uterus contains white pus.

Arthritis: Cloudy fluid and pus present in the leg joints

Fig. 211: Pasteurellosis. Yellowish-white fibrinous deposits in the abdominal cavity.

Judgement : Carcass of the animal is *condemned.*

- if clinical signs of severe acute pneumonia or peritonitis with accompanied fever are manifested on postmortem with swollen haemorrhagic lungs or fibrinous deposits on the peritoneum and organs.
- if multiple abscesses are found throughout the body or in the abdominal cavity.
- in cases of septicemia on antemortem and postmortem examination
- if inflammation of the joints is associated with emaciation

A mild form of the disease showing colds, snuffles, middle ear infection, mastitis or metritis, which do not affect the wholesomeness of the meat or cause systemic changes, may have a *favourable judgement* of the carcass. A few well off abscesses may also render meat fit for human consumption although the carcass may be judged *inferior* due to mutilation caused by removing of abscesses. Consumer should be made aware of this defect by the controlling authority.

Differential diagnosis : Salmonellosis and coccidiosis. Bacteria such as E. coli, Pseudomonas, Listeria and Proteus may cause metritis in rabbits. Staphylococcus aureus has been cultured from mastitis, metritis and arthritis cases. Staphylococcus and Bordetella have been isolated from the respiratory lesions and Streptococcus spp and Actinomyces pyogenes from abscesses.

Parasitic diseases

Diseases caused by helminths

Tape worm larvae in rabbits

Tape worm infection in rabbits (intermediate host) and dogs and cats (definitive host) is relatively harmless. However, in its severe form may cause chronic inflammation of the intestine, emaciation or intestinal obstruction. Infection in rabbits is of greater importance because the larval stages of the parasite develop in different organs and the musculature and often cause decreased food utilization and wasting of the host. The carcass or portion of the carcass may also be unfit for human consumption in severe cases which further contributes to the economic loss.

Cysticercus pisiformis is the cystic stage of *Taenia pisiformis* which occurs in dogs and rarely in cats. The larval stage develops in rodents, particularly rabbit and hare. The mature larvae are found in the peritoneal cavity and frequently in the mesentery of rabbits (Fig. 212). The cysts are the size of a pea, hence the name C. pisiformis. They are filled with clear fluid in early stages. There is formation of pus in older lesions.

Fig. 212: Tapeworm larvae in rabbits. Cysticercus pisiformis in the mesentery of rabbit.

Cysticercus fasciolaris is the cystic stage of *Taenia taeniaeformis*, known as the cat tapeworm. This parasite can be recognized by the lack of a neck and the bell-shaped posterior segments. The intermediate host are the rabbit, rat, mouse and other rodents. Cysticercus fasciolaris is 2.5 cm long and develops in the liver.

Coenurus serialis is the intermediate stage of *Taenia serialis*, a tapeworm of the dog and fox. Coenurus serialis is almost fatal to hares, rabbits and other related rodents. It is passed into the connective tissue of the lumbar muscles, hind leg muscles and rarely into the jaw muscles. Coenurus serialis may also occur accidentally in the brain and muscle tissue of humans. The mature cyst in rabbits is ovoid or round, approximately 5 cm in diameter and has scolices the size of a rice grain. It may have as many as forty scolices. Sometimes these Coenuri behave like hydatids by budding off new cysts internally or externally. These internal cysts are frequently fertile. The external cysts are attached by stalks and are often sterile. They resemble a bunch of grapes.

Judgement : Rabbit carcases with extensive *Cysticercus pisiformis* infestation associated with cheesy material in older lesions and accompanied with emaciation are *condemned*. Slight or moderate infestation in which cysts contain straw coloured fluid, may have a *favourable judgement*. In *Coenurus serialis* infestation, the rabbit carcass is *approved* if only few cysts are present in the musculature. In extensive infestation the carcass is *condemned*.

Diseases caused by protozoa

Coccidiosis

Coccidiosis is the most common parasitic disease of rabbits which occurs in hepatic and intestinal forms. Liver coccidiosis is caused by *Eimeria stiedae* and intestinal coccidiosis by *E. magna, E. perforans* and *E. irresidua.*

Transmission : Faecal contamination of water and food containing oocysts. Humid, dirty and overcrowded rabbit houses predispose rabbits to the infection.

Antemortem findings :

1. Loss of appetite and emaciation
2. Anaemia
3. Diarrhoea in terminal stage
4. Dry fur, pot belly and death

Postmortem findings :

1. Small greyish white nodules in the liver in E. stiedae infections (Fig. 213)
2. Older lesions coalesce and form cheesy masses
3. In intestinal coccidiosis the contents of the intestine are soft and the lesions pinhead size.
4. Greyish white flakes in the intestinal wall
5. Thickened and pale intestinal wall in more advanced cases
6. Oocysts present in the intestinal content

Judgement : Carcass in good flesh is *approved.* If the disease is associated with emaciation, the carcass is *condemned.*

Differential diagnosis : Pasteurellosis, tuberculosis, pseudotuberculosis, listeriosis and salmonellosis.

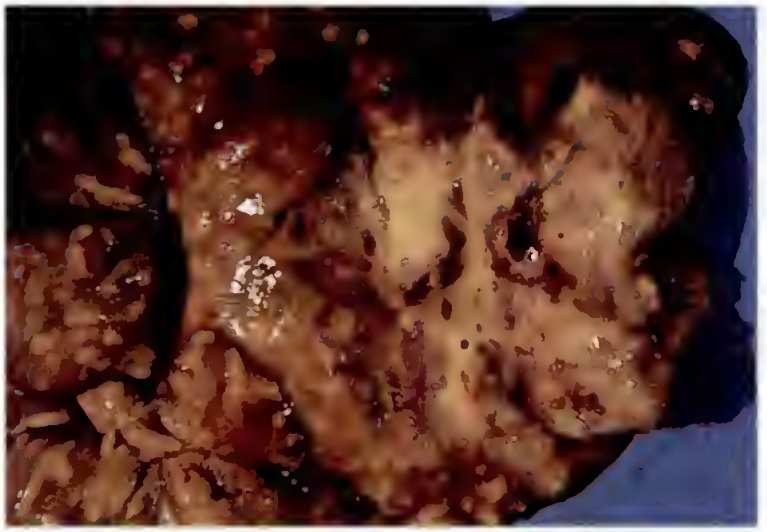


Fig. 213: Coccidiosis. Enlarged liver with multifocal greyish-white coalescing lesions and yellowish liquid pus caused by E. stidae.

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Meat inspection is the sanitary control of slaughter animals and meat, and its aim is to provide safe and wholesome meat for human consumption. In many developing regions and in particular in rural abattoirs, meat inspectors often lack the necessary information and guidelines to assess the sanitary status of carcasses, meat and organs from slaughter animals. FAO has therefore endeavoured to prepare concise guidelines on the subject together with colour illustrations demonstrating the pathological lesions that may occur in bovines, small ruminants, pigs, game, poultry and rabbits. The book contains contributions from experts from different livestock-producing areas in developed and developing countries. It is intended to assist meat inspectors, particularly in developing regions, and to serve as a training manual for meat inspection courses.

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